09/ 891,671

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TERMINAL (ENTER 1, 2, 3, OR ?):2

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 NEWS  1          Web Page URLs for STN Seminar Schedule - N. America
 NEWS  2  Apr 08  "Ask CAS" for self-help around the clock
 NEWS  3  Apr 09  BEILSTEIN: Reload and Implementation of a New Subject Area
 NEWS  4  Apr 09  ZDB will be removed from STN
 NEWS  5  Apr 19  US Patent Applications available in IFICDB, IFIPAT, and IFIUDB
 NEWS  6  Apr 22  Records from IP.com available in CAPLUS, HCAPLUS, and ZCAPLUS
 NEWS  7  Apr 22  BIOSIS Gene Names now available in TOXCENTER
 NEWS  8  Apr 22  Federal Research in Progress (FEDRIP) now available
 NEWS  9  Jun 03  New e-mail delivery for search results now available
 NEWS 10  Jun 10  MEDLINE Reload
 NEWS 11  Jun 10  PCTFULL has been reloaded
 NEWS 12  Jul 02  FOREGE no longer contains STANDARDS file segment
 NEWS 13  Jul 22  USAN to be reloaded July 28, 2002;
                  saved answer sets no longer valid
 NEWS 14  Jul 29  Enhanced polymer searching in REGISTRY
 NEWS 15  Jul 30  NETFIRST to be removed from STN
 NEWS 16  Aug 08  CANCERLIT reload
 NEWS 17  Aug 08  PHARMAMarketLetter(PHARMAML) - new on STN
 NEWS 18  Aug 08  NTIS has been reloaded and enhanced
 NEWS 19  Aug 09  JAPIO to be reloaded August 25, 2002
 NEWS 20  Aug 19  Aquatic Toxicity Information Retrieval (AQUIRE)
                  now available on STN
 NEWS 21  Aug 19  IFIPAT, IFICDB, and IFIUDB have been reloaded
 NEWS 22  Aug 19  The MEDLINE file segment of TOXCENTER has been reloaded
 NEWS 23  Aug 26  Sequence searching in REGISTRY enhanced

 NEWS EXPRESS  February 1 CURRENT WINDOWS VERSION IS V6.0d,
               CURRENT MACINTOSH VERSION IS V6.0a(ENG) AND V6.0Ja(JP),
               AND CURRENT DISCOVER FILE IS DATED 05 FEBRUARY 2002
 NEWS HOURS    STN Operating Hours Plus Help Desk Availability
 NEWS INTER    General Internet Information
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 NEWS PHONE    Direct Dial and Telecommunication Network Access to STN
 NEWS WWW      CAS World Wide Web Site (general information)
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FILE 'HOME' ENTERED AT 17:22:47 ON 02 SEP 2002

09/ 891,671

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=> file reg
COST IN U.S. DOLLARS                                    SINCE FILE      TOTAL
                                                           ENTRY    SESSION
FULL ESTIMATED COST                                         0.21       0.21

FILE 'REGISTRY' ENTERED AT 17:22:55 ON 02 SEP 2002
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DICTIONARY FILE UPDATES:   1 SEP 2002  HIGHEST RN 446010-91-9

TSCA INFORMATION NOW CURRENT THROUGH MAY 20, 2002

  Please note that search-term pricing does apply when
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Calculated physical property data is now available.  See HELP PROPERTIES
for more information.  See STNote 27, Searching Properties in the CAS
Registry File, for complete details:
http://www.cas.org/ONLINE/STN/STNOTES/stnotes27.pdf

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L1      STRUCTURE UPLOADED

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L1 HAS NO ANSWERS
L1              STR
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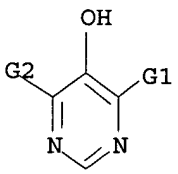


```
G1 H,N
G2 H,Ak


Structure attributes must be viewed using STN Express query preparation.


=> s l1
SAMPLE SEARCH INITIATED 17:23:16 FILE 'REGISTRY'
SAMPLE SCREEN SEARCH COMPLETED -   4046 TO ITERATE

 24.7% PROCESSED    1000 ITERATIONS                              3 ANSWERS
INCOMPLETE SEARCH (SYSTEM LIMIT EXCEEDED)
SEARCH TIME: 00.00.01

FULL FILE PROJECTIONS:  ONLINE  **COMPLETE**
                        BATCH   **COMPLETE**
PROJECTED ITERATIONS:        77108 TO    84732
PROJECTED ANSWERS:              33 TO      451

L2            3 SEA SSS SAM L1
```

09/ 891,671

=> s l1 ful
FULL SEARCH INITIATED 17:23:21 FILE 'REGISTRY'
FULL SCREEN SEARCH COMPLETED - 81672 TO ITERATE

100.0% PROCESSED 81672 ITERATIONS 330 ANSWERS
SEARCH TIME: 00.00.12

L3 330 SEA SSS FUL L1

=> file caplus
COST IN U.S. DOLLARS SINCE FILE ENTRY TOTAL SESSION
FULL ESTIMATED COST 140.28 140.49

FILE 'CAPLUS' ENTERED AT 17:23:39 ON 02 SEP 2002
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FILE COVERS 1907 - 2 Sep 2002 VOL 137 ISS 10
FILE LAST UPDATED: 1 Sep 2002 (20020901/ED)

This file contains CAS Registry Numbers for easy and accurate substance identification.

CAS roles have been modified effective December 16, 2001. Please check your SDI profiles to see if they need to be revised. For information on CAS roles, enter HELP ROLES at an arrow prompt or use the CAS Roles thesaurus (/RL field) in this file.

=> s l3
L4 323 L3

=> s electron donating
1115493 ELECTRON
13528 DONATING
L5 9814 ELECTRON DONATING
(ELECTRON(W)DONATING)

=> s l4 and l5
L6 1 L4 AND L5

=> d l6 ibib abs fhitstr

L6 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1984:472692 CAPLUS
DOCUMENT NUMBER: 101:72692
TITLE: Syntheses of some pyrimidine N-oxides
AUTHOR(S): Jovanovic, Misa V.
CORPORATE SOURCE: Dep. Chem., South. Methodist Univ., Dallas, TX, 75275, USA
SOURCE: Can. J. Chem. (1984), 62(6), 1176-80

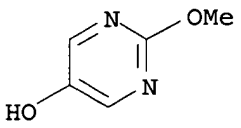
09/ 891,671

CODEN: CJCHAG; ISSN: 0008-4042
DOCUMENT TYPE: Journal
LANGUAGE: English
OTHER SOURCE(S): CASREACT 101:72692

AB Various monosubstituted pyrimidines and methylpyrimidines were N-oxidized with a no. of different peracids. In general, they are more susceptible to side reactions accompanying N-oxidn. than other .pi.-deficient diazines and triazines. Unsym. pyrimidines, which can potentially yield 2 isomeric products, were N-oxidized preferentially at the site para to strong **electron-donating** substituents. Weaker ring-activating groups, such as Me, are mainly ortho-directing and only aid in the N-oxidn. of pyrimidine nuclei having ortho/para-directing substituents.

IT **91233-70-4P**
RL: FORM (Formation, nonpreparative); PREP (Preparation)
(formation of, in N-oxidn. of pyrimidine analogs)

RN 91233-70-4 CAPLUS
CN 5-Pyrimidinol, 2-methoxy- (9CI) (CA INDEX NAME)




```
=> s inhibit? and oxidation
       1503154 INHIBIT?
        363690 OXIDATION
L7       24310 INHIBIT? AND OXIDATION

=> d his

     (FILE 'HOME' ENTERED AT 17:22:47 ON 02 SEP 2002)

     FILE 'REGISTRY' ENTERED AT 17:22:55 ON 02 SEP 2002
L1               STRUCTURE UPLOADED
L2             3 S L1
L3           330 S L1 FUL

     FILE 'CAPLUS' ENTERED AT 17:23:39 ON 02 SEP 2002
L4           323 S L3
L5          9814 S ELECTRON DONATING
L6             1 S L4 AND L5
L7         24310 S INHIBIT? AND OXIDATION

=> s l4 and l7
L8             0 L4 AND L7

=> s l7 and pyrimidin?
         62774 PYRIMIDIN?
L9          73 L7 AND PYRIMIDIN?

=> s l9 and hydroxy
        307940 HYDROXY
L10         14 L9 AND HYDROXY

=> s l7 and (hydroxy same pyrimidin?)
        307940 HYDROXY
       1176079 SAME
         62774 PYRIMIDIN?
             0 HYDROXY SAME PYRIMIDIN?
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                (HYDROXY(W)SAME(W)PYRIMIDIN?)
L11          0 L7 AND (HYDROXY SAME PYRIMIDIN?)

=> s l7 and (hydroxy pyrimidin?)
        307940 HYDROXY
         62774 PYRIMIDIN?
           102 HYDROXY PYRIMIDIN?
                (HYDROXY(W)PYRIMIDIN?)
L12          0 L7 AND (HYDROXY PYRIMIDIN?)

=> d l9 1- ibib abs fhitstr
YOU HAVE REQUESTED DATA FROM 73 ANSWERS - CONTINUE? Y/(N):y

L9   ANSWER 1 OF 73  CAPLUS  COPYRIGHT 2002 ACS
ACCESSION NUMBER:        2001:858580  CAPLUS
DOCUMENT NUMBER:         136:145186
TITLE:                   Comparison of DNA damage photoinduced by ketoprofen,
                         fenofibric acid and benzophenone via electron and
                         energy transfer
AUTHOR(S):               Lhiaubet, Virginie; Paillous, Nicole; Chouini-Lalanne,
                         Nadia
CORPORATE SOURCE:        Laboratoire des Interactions Moleculaires et
                         Reactivite Chimique et Photochimique, Universite Paul
                         Sabatier, Toulouse, Fr.
SOURCE:                  Photochemistry and Photobiology (2001), 74(5), 670-678
                         CODEN: PHCBAP; ISSN: 0031-8655
PUBLISHER:               American Society for Photobiology
DOCUMENT TYPE:           Journal
LANGUAGE:                English
AB   Ketoprofen (KP) and fenofibrate, resp., anti-inflammatory and
     hypolipidemic agents, promote anormal photosensitivity in patients and may
     induce photoallergic cross-reactions correlated to their benzophenone-like
     structure.  Here, their ability to photosensitize the degrdn. of biol.
     targets was investigated in DNA.  The photosensitization of DNA damage by
     KP and fenofibric acid (FB), the main metabolite of fenofibrate, and their
     parent compd., benzophenone (BZ), was examd. on a 32P-end-labeled
     synthetic oligonucleotide in phosphate-buffered soln. using gel sequencing
     expts.  Upon irradn. at .lambda. > 320 nm, piperidine-sensitive lesions
     were induced in single-stranded oligonucleotides by KP, FB and BZ at all G
     sites to the same extent.  This pattern of damage, enhanced in D2O is
     characteristic of a Type-II mechanism.  Spin trapping expts. using
     2,2,6,6-tetramethyl-4-piperidone have confirmed the prodn. of singlet
     oxygen during drug photolysis.  On double-stranded oligonucleotides,
     highly specific DNA break occurred selectively at 5'-G of a 5'-GG-3'
     sequence, after alkali treatment.  Prolonged irradn. led to the degrdn. of
     all G residues, with efficiency decreasing in the order 5'-GG > 5'-GA >
     5'-GC > 5'-GT, in good agreement with the calcd. lowest ionization
     potentials of stacked nucleobase models supporting the assumption of a
     Type-I mechanism involving electron transfer, also obsd. to a lesser
     extent with adenine.  Cytosine sites were also affected but the action of
     mannitol which selectively inhibited cytosine lesions suggests,
     in this case, the involvement of hydroxyl radical, also detected by
     electronic paramagnetic resonance using 5,5-dimethyl-1-pyrrolidine-1-oxide
     as spin trap.  On a double-stranded 32P-end-labeled 25-mer oligonucleotide
     contg. TT and TTT sequences, the three compds. were found to
     photosensitize by triplet-triplet energy transfer the formation of
     cyclobutane thymine dimers detected using T4 endonuclease V.
REFERENCE COUNT:         50    THERE ARE 50 CITED REFERENCES AVAILABLE FOR THIS
                               RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9   ANSWER 2 OF 73  CAPLUS  COPYRIGHT 2002 ACS
ACCESSION NUMBER:        2001:394691  CAPLUS
DOCUMENT NUMBER:         135:166578
```

09/ 891,671

TITLE: Mechanistic investigations of **oxidation** of purine and **pyrimidine** base components of nucleic acids by bromamine-B in aqueous alkaline medium: A kinetic approach
AUTHOR(S): Vaz, Nirmala; Puttaswamy
CORPORATE SOURCE: Department of Chemistry, Central College, Bangalore University, Bangalore, 560 001, India
SOURCE: Studies in Surface Science and Catalysis (2001), 133(Reaction Kinetics and the Development and Operation of Catalytic Processes), 495-500
CODEN: SSCTDM; ISSN: 0167-2991
PUBLISHER: Elsevier Science B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Mechanism of oxidn. of purine bases (adenine and guanine) and **pyrimidine** bases (uracil, thymine and cytosine) in presence of NaOH by bromamine-B(BAB) was studied. The reactions follow identical kinetics for all the bases, being first order dependence on [BAB]o and fractional order each in [substrate]o and [NaOH]. Addn. of the reaction product retards the rate and the dielec. effect is pos. Variation of ionic strength and addn. of halide ions had no effect on the rate. Proton inventory studies were made in H2O-D2O mixts. for adenine and cytosine. Oxidn. products were identified and activation parameters were evaluated. An isokinetic relation is obsd. with .beta. = 336 K indicated that enthalpy factors control the rate. The rate of oxidn. of purines is in the order: guanine > adenine while in case of **pyrimidines** the order is thymine > uracil > cytosine. A suitable mechanism is proposed and discussed.

REFERENCE COUNT: 16 THERE ARE 16 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 3 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:279860 CAPLUS
DOCUMENT NUMBER: 135:62958
TITLE: The Chemical Development of CI-972 and CI-1000: A Continuous Nitration, A MgCl2/Et3N-Mediated C-Alkylation of a Chloronitropyrimidine, A Catalytic Protodediazotization of a Diazonium Salt, and an Air **Oxidation** of an Amine
AUTHOR(S): De Jong, Randall L.; Davidson, James G.; Dozeman, Gary J.; Fiore, Philip J.; Giri, Punam; Kelly, Margaret E.; Puls, Timothy P.; Seamans, Ronald E.
CORPORATE SOURCE: Pfizer Global Research and Development Holland Laboratories, Holland, MI, 49424, USA
SOURCE: Organic Process Research & Development (2001), 5(3), 216-225
CODEN: OPRDFK; ISSN: 1083-6160
PUBLISHER: American Chemical Society
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Efficient, large-scale processes were developed for the prepn. of the potent PNP **inhibitors**: 2,6-diamino-3,5-dihydro-7-(3-thienylmethyl)-4H-pyrrolo[3,2-d]**pyrimidin**-4-one hydrochloride monohydrate and 2-amino-3,5-dihydro-7-(3-thienylmeth- yl)-4H-pyrrolo[3,2-d]**pyrimidin**-4-one hydrochloride monohydrate (I). We report (1) a safe, continuous nitration process for the prepn. of 2-amino-6-chloro-5-nitro-4-**pyrimidinol** and its stable diisopropylamine salt, (2) the first MgCl2/Et3N-mediated C-alkylation of a chloronitropyrimidine, (3) a rare catalytic protodediazotization of 2-amino-4-oxo-7-thiophen-3-ylmethyl-4,5-dihydro-3H-pyrrolo[3,2-d] **pyrimidine**-6-diazonium chloride, (4) a single-step process to prep. I directly from 2-amino-6-hydroxy-5-nitro-.alpha.-(3-thienylmethyl)-4-**pyrimidineacetonitrile** using a sponge nickel-catalyzed redn.,

and (5) a method to convert the over-redn. byproduct: 2,5-diamino-6-(1-aminomethyl-2-thiophen-3-yl-ethyl)-**pyrimidin**-4-ol into I using air oxidn.
REFERENCE COUNT: 32 THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 4 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:107651 CAPLUS
DOCUMENT NUMBER: 134:279593
TITLE: Synthesis of chiral pharmaceutical intermediates by oxidoreductases
AUTHOR(S): Patel, Ramesh N.; Hanson, Ronald L.
CORPORATE SOURCE: Department of Enzyme Technology, Process Research, Bristol-Myers Squibb Pharmaceutical Research Institute, New Brunswiick, NJ, 08903, USA
SOURCE: ACS Symposium Series (2001), 776(Applied Biocatalysis in Specialty Chemicals and Pharmaceuticals), 216-247
CODEN: ACSMC8; ISSN: 0097-6156
PUBLISHER: American Chemical Society
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review with 77 refs. Chiral intermediates were prepd. by enzymic process using oxidoreductases for the chem. synthesis of pharmaceutical drug candidates. These includes: the microbial redn. of 1-(4-fluorophenyl)-4-[4-(5-fluoro-2-**pyrimidinyl**)-1-piperazinyl]-1-butanone to R-(+)-1-(4-fluoro-phenyl)-4-[4-(5-fluoro-2-**pyrimidinyl**)-1-piperazinyl]-1-butanol [R-(+)-BMY 14802], an antipsychotic agent; the redn. of N-(4-(1-oxo-2-chloroacetyl ethyl) Ph methane) sulfonamide to corresponding chiral alc. , an intermediate for D-(+)-N-[4-[1-Hydroxy-2-[(-methylethyl)amino]ethyl]phenyl] methanesulfonamide [D-(+) sotalol], a .beta.-blocker with class III antiarrhythmic properties; biotransformation of N-.epsilon.-carbobenzoxy (CBZ)-L-lysine 7 to CBZ-L-oxylysine, an intermediate needed for synthesis of (S)-1-[6-amino-2-[[hydroxy(4-phenylbutyl) phosphinyl]oxy]1-oxohexyl]-L-proline [ceronapril], a new angiotensin converting enzyme [ACE] **inhibitor**;, enzymic synthesis L-.beta.-hydroxyvaline from .alpha.-keto-.beta.-hydroxy isovalerate. L-.beta.-Hydroxy valine is a key chiral intermediate needed for the synthesis of [S-(Z)]-[[[1-(2-Amino-4-thiazolyl)-2- [[2,2-dimethyl-4-oxo-1-(sulfooxy)-3-azetidinyl] amino]-2-oxoethylidene] amino]oxy]acetic acid [tigemonam], a orally active monobactam, enzymic synthesis of L-6-hydroxynorleucine, and enzymic synthesis of (S)-2-amino-5-(1,3-dioxolan-2-yl)-pentanoic acid (allysine ethylene acetal), one of three building blocks used for an alternative synthesis of omapatrilat, a vasopeptidase **inhibitor**.
REFERENCE COUNT: 77 THERE ARE 77 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

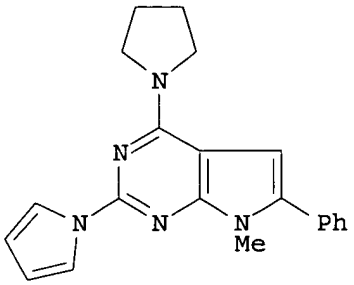
L9 ANSWER 5 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1998:648906 CAPLUS
DOCUMENT NUMBER: 130:20319
TITLE: Structural and functional comparison of agents interfering with dihydroorotate, succinate and NADH **oxidation** of rat liver mitochondria
AUTHOR(S): Jockel, Johannes; Wendt, Bernd; Loffler, Monika
CORPORATE SOURCE: Institute for Physiological Chemistry, School of Medicine, Philipps-University, Marburg, D-35033, Germany
SOURCE: Biochemical Pharmacology (1998), 56(8), 1053-1060
CODEN: BCPCA6; ISSN: 0006-2952
PUBLISHER: Elsevier Science Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Mitochondrially bound dihydroorotate dehydrogenase (EC 1.3.99.11)

catalyzes the fourth sequential step in the de novo synthesis of uridine monophosphate; this enzyme uses ubiquinone as the proximal and cytochrome oxidase as is the ultimate electron transfer system. Here, seven compds. with proven antiproliferative activity and in vitro antipyrimidine effects were investigated with isolated functional mitochondria of rat tissues in order to differentiate their anti-dihydroorotate dehydrogenase potency vs. putative effects on the respiratory chain enzymes. Ten .mu.M of brequinar sodium, the leflunomide derivs. A77-1726, [2-cyano-3-cyclopropyl-3-hydroxy-enoic acid (4-trifluoromethyl)-amide], MNA 279, (2-cyano-N-(4-cyanophenyl-3-cyclopropyl-3-oxo-propanamide), MNA715 (2-cyano-3-hydroxy-N-4-(trifluoromethyl)-phenyl-6-heptanamide), HR325 (2-cyano-3-cyclopropyl-3-hydroxy-N-[3'-methyl-4'-(trifluoromethyl)phenyl]-propenamide), and the diazine toltrazuril completely **inhibited** the dihydroorotate-induced oxygen consumption of liver mitochondria. Succinate and NADH oxidn. were found to be influenced only at elevated drug concn. (100 .mu.M), with the exception of HR325, 10 .mu.M of which caused a 70% **inhibition** of NADH and 50% **inhibition** of succinate oxidn. This was comparable to the effects of toltrazuril, which caused an approx. 75% **inhibition** of NADH oxidn. Ciprofloxacin was shown here to have only marginal effects on the redox activities of the inner mitochondrial membrane. This differentiation of drug effects on mitochondrial functions will contribute to a better understanding of the in vivo pharmacol. activity of these drugs, which are presently in clin. trials because of their immunosuppressive, cytostatic or anti-parasitic activity. A comparison of the influence of A77-1726, HR325, brequinar and 2,4-dinitrophenol on energetically coupled rat liver mitochondria revealed only a weak uncoupling potential of A77-1726 and brequinar. In addn., a modeling study was raised to search for common spatial arrangements of functional groups essential for binding of **inhibitors** to dihydroorotate dehydrogenase. From the structural comparison of different metabolites and **inhibitors** of **pyrimidine** metab., a 6-point model was obtained by conformational anal. for the drugs tested on mitochondrial functions, pharmacophoric perception and mapping. We propose our model in combination with kinetic data for a rational design of highly specific **inhibitors** of dihydroorotate dehydrogenase.

REFERENCE COUNT: 34 THERE ARE 34 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 6 OF 73 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1998:622209 CAPLUS
DOCUMENT NUMBER: 129:343459
TITLE: Chemical **Oxidation** of 2,4-Diaminopyrrolo[2,3-d]**pyrimidines**
AUTHOR(S): Bundy, Gordon L.; Gremban, Robert S.; Banitt, Lee S.; Palmer, John R.; Mizsak, Stephen A.; Han, Fusen
CORPORATE SOURCE: Medicinal Chemistry and Structural Analytical and Medicinal Chemistry, Pharmacia Upjohn Company, Kalamazoo, MI, 49001-0199, USA
SOURCE: Journal of Organic Chemistry (1998), 63(21), 7542-7546
CODEN: JOCEAH; ISSN: 0022-3263
PUBLISHER: American Chemical Society
DOCUMENT TYPE: Journal
LANGUAGE: English
GI



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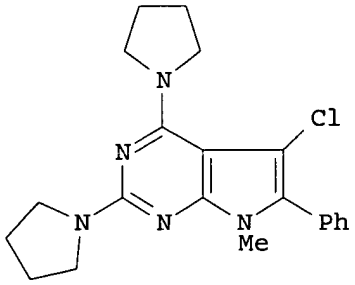


II

AB Oxidn. of lipophilic antioxidant PNU-87663 by a variety of nonbiol. oxidizing agents was investigated. E.g., stirring CHCl3 solns. of PNU-87663 in air for 1 wk gave small amts. of I and II. None of the oxidn. products retained significant levels of lipid peroxidn. **inhibiting** activity.

L9 ANSWER 7 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1998:621121 CAPLUS
DOCUMENT NUMBER: 129:239916
TITLE: Therapeutic augmentation of oxyalkylene diesters and butyric acid derivatives with **inhibitors** of fatty acid .beta.-**oxidation**
INVENTOR(S): Rephaeli, Ada
PATENT ASSIGNEE(S): Beacon Laboratories, L.L.C., USA
SOURCE: PCT Int. Appl., 58 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9840078	A1	19980917	WO 1998-US4652	19980311
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
US 5939455	A	19990817	US 1997-814222	19970311
AU 9865478	A1	19980929	AU 1998-65478	19980311

PRIORITY APPLN. INFO.: US 1997-814222 19970311
WO 1998-US4652 19980311

AB This invention provides a method of augmenting the therapeutic activity of an oxyalkylene-contg. compd., butyric acid, a butyric acid salt or butyric acid deriv. by administering an **inhibitor** of .beta.-oxidn. of fatty acids to a patient or to host cells. Pharmaceutical compns. are also included.

L9 ANSWER 8 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1998:186491 CAPLUS
DOCUMENT NUMBER: 128:239464
TITLE: Determination of prodrugs metabolizable by the liver and therapeutic use thereof
INVENTOR(S): Cheng, Yung-Chi; Chang, Chien-Neng
PATENT ASSIGNEE(S): Yale University, USA

09/ 891,671

SOURCE: U.S., 24 pp., Cont.-in-part of U.S. Ser. No. 701,462, abandoned.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 5728684	A	19980317	US 1994-146164	19940419
ZA 9203495	A	19930331	ZA 1992-3495	19920514
WO 9220816	A1	19921126	WO 1992-US4142	19920515
W: AT, AU, BB, BG, BR, CA, CH, CS, DE, DK, ES, FI, GB, HU, JP, KP, KR, LK, LU, MG, MN, MW, NL, NO				
RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, DE, DK, ES, FR, GA, GB, GN, GR, IT, LU, MC, ML, MR, NL, SE				
IL 121375	A1	19981206	IL 1992-121375	19920515

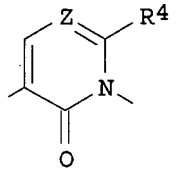
PRIORITY APPLN. INFO.: US 1991-701462 B2 19910515
US 1992-829474 B2 19920203
WO 1992-US4142 W 19920515
IL 1992-101879 A3 19920515
OTHER SOURCE(S): MARPAT 128:239464

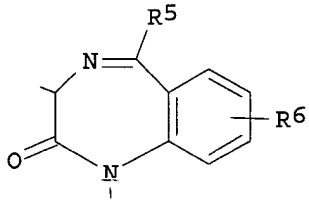
AB A method of ascertaining if a prodrug is useful for treating a disease is disclosed. The prodrug is acceptable if it is metabolized in liver cells by aldehyde oxidase to produce an active drug or metabolite. Prodrugs are shown equally effective in treating diseases as the active drug itself with many benefits and without as many assocd. side effects. Methods for treating cancers with e.g. 5-iodo-2-**pyrimidinone**-deoxyribose are also described.

L9 ANSWER 9 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1998:126655 CAPLUS
DOCUMENT NUMBER: 128:192666
TITLE: Preparation of acetamides, their use as chymase **inhibitors** and angiotensin II **inhibitors**, and cardiovascular agents containing them
INVENTOR(S): Akaha, Atsushi; Takenaka, Kohei; Itani, Hiromichi; Sato, Akihiro; Nakanishi, Isao
PATENT ASSIGNEE(S): Fujisawa Pharmaceutical Co., Ltd., Japan
SOURCE: Jpn. Kokai Tokkyo Koho, 12 pp.
CODEN: JKXXAF
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 10053579	A2	19980224	JP 1997-160803	19970618

PRIORITY APPLN. INFO.: AU 1996-626 19960624
OTHER SOURCE(S): MARPAT 128:192666
GI

Q^1 = 

Q^2 = 

AB R1NHXYCONHCHR2COR3 I [R1 = H, protecting group; R2 = ar(lower)alkyl; R3 = lower haloalkyl, (protected) CO2H; X = Q1, Q2; R4, R5 = halo-, lower alkoxy-, or Ph-substituted aryl, cyclo(lower)alkyl; R6 = H, lower alkyl; Z = N, CH; Y = lower alkylene] or their salts, useful for prevention or treatment of heart and/or circulation disorders, are prepd. by oxidn. of R1aNHXYCONHCHR2CHR3OH (R1a = protecting group; R2, R3, X, Y = same as above) or their salts, followed by optional deprotection. Oxidn. of 905 mg 2-[5-[(benzyloxycarbonyl)amino]-2-(4-fluorophenyl)-1,6-dihydro-6-oxo-1-**pyrimidinyl**]-N-[2-(4,4,4-trifluoro-3-hydroxy-1-phenyl)butyl]acetamide with 1,1,1-triacetoxy-1,1-dihydro-1,2-benziodoxol-1-(1H)-one at room temp. for 15 h in CH2Cl2 gave 644 mg the corresponding ketone deriv., which **inhibited** chymase at IC50 of <1.0 .times. 10-5 M.

L9 ANSWER 10 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1997:44786 CAPLUS
DOCUMENT NUMBER: 126:63808
TITLE: Aqueous bath with organosilanes and **oxidation inhibitors** for treatment of copper surface to promote laminate bonding and soldering
INVENTOR(S): Aoyama, Masayuki; Morita, Ryoji; Kawaguchi, Jyun
PATENT ASSIGNEE(S): Henkel Corporation, USA; Aoyama, Masayuki; Morita, Ryoji; Kawaguchi, Jyun
SOURCE: PCT Int. Appl., 24 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9636747	A1	19961121	WO 1996-US6549	19960514
W: CA, US				
JP 08311658	A2	19961126	JP 1995-142656	19950517
US 5925174	A	19990720	US 1997-930080	19971114

PRIORITY APPLN. INFO.: JP 1995-142656 19950517
WO 1996-US6549 19960514
OTHER SOURCE(S): MARPAT 126:63808
AB The aq. bath for coating treatment of Cu or cu-alloy surface contains: (a) dissolved and/or dispersed org. solvent; (b) an organosilane coupling agent having functional vinyl, mercapto, amino, and/or glycidyloxy

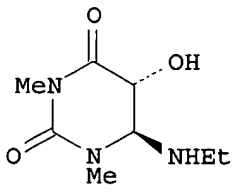
moieties; and (c) Cu-oxidn. **inhibitor** selected from azoles, azines, arom. secondary amines, and/or arom. diacyl hydrazide compds. The aq. baths contain the org. solvent at 0.01-15, organosilane at 0.01-30, and the **inhibitor** at 0.01-5% with the organosilane/ **inhibitor** ratio of (2-8):1, and are suitable for the treatment of Cu foils to improve laminate bonding and soldering for elec. printed circuits. The cleaned Cu foils 35 .mu.m thick can be dip coated for 20 s in the aq. bath contg. 3-(glycidyloxy)propyltrimethoxysilane 0.08, 2-methylimidazole 0.02, and MeOH 10%, followed by drying with hot air at 100.degree.. The treatment provides resistance to Cu migration in close-spaced elec. printed circuits, resistance to thermal damage by molten solder, and increased adhesion strength (esp. at .apprx.150.degree.) in laminates.

L9 ANSWER 11 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1996:629081 CAPLUS
DOCUMENT NUMBER: 125:267783
TITLE: Potent peroxisome proliferators **inhibit** .beta.-**oxidation** in the isolated perfused rat liver
AUTHOR(S): Bojes, Heidi K.; Thurman, Ronald G.
CORPORATE SOURCE: Dept. Pharmacolo., University North Carolina Chapel Hill, NC, 27599-7365, USA
SOURCE: Toxicology and Applied Pharmacology (1996), 140(2), 322-327
CODEN: TXAPA9; ISSN: 0041-008X
PUBLISHER: Academic
DOCUMENT TYPE: Journal
LANGUAGE: English

AB It is unknown whether peroxisome proliferators decrease hepatic fatty acid oxidn. via uncoupling of respiration or if they **inhibit** extramitochondrial fatty acyl CoA synthesis. Therefore, the purpose of this study was to examine both processes simultaneously using the isolated perfused liver, a whole cell prepn. where enzymes and biochem. processes can be monitored continuously under nearly physiol. conditions. Accordingly, ketone body formation (.beta.-hydroxybutyrate + acetoacetate) from lipid metab. and oxygen uptake, which is increased by uncoupling agents, were monitored at the same time. 2-Bromooctanoate, a known **inhibitor** of acyl CoA synthetase, decreased ketone body formation in a dose-dependent manner without altering cellular respiration (half-maximal **inhibition**, .apprx.25 .mu.M) and concomitantly increased protein kinase C nearly fourfold also in a dose-dependent fashion. Ketogenesis was also blocked maximally 50-66% with mono(ethylhexyl) phthalate, 4-chloro-6-(2,3-xylidino)-2-**pyrimidinylthioacetic** acid (WY-14,643), and nafenopin, potent peroxisome proliferators and tumor promoters. These compds. also increased protein kinase C three- to fourfold without altering oxygen uptake significantly. Thus, lipid metab. appears to be the prime target of potent peroxisome proliferators most likely on actions via acyl CoA synthetase rather than oxidative phosphorylation. In contrast, weak peroxisome proliferators and tumor promoters, di(ethylhexyl) phthalate and 2-ethylhexanol, did not affect ketogenesis, oxygen consumption, or protein kinase C at similar concns. Addnl., octanoate increased ketone body formation in the presence of nafenopin. Because octanoate is metabolized by mitochondrial acyl CoA synthetase independent of carnitine acyltransferase, these results indicate that the nafenopin does not **inhibit** mitochondrial .beta.-oxidn. Take together, it is concluded that potent peroxisome proliferators preferentially block ketogenesis without altering cellular respiration in the liver. This phenomenon, which occurs due to **inhibition** of acyl CoA synthetase, leads to an elevation of free fatty acids that stimulates protein kinase C and promotes cell proliferation.

L9 ANSWER 12 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1996:210224 CAPLUS
DOCUMENT NUMBER: 124:306408
TITLE: Effect of various potential **inhibitors**, activators and inducers on the N-**oxidation** of isomeric aromatic diazines in vitro using rabbit liver microsomal preparations
AUTHOR(S): Altuntas, T. G.; Gorrod, J. W.
CORPORATE SOURCE: Chelsea Dep. of Pharmacy, Univ. of London, London, SW3 6LX, UK
SOURCE: Xenobiotica (1996), 26(1), 9-15
CODEN: XENOBH; ISSN: 0049-8254
PUBLISHER: Taylor & Francis
DOCUMENT TYPE: Journal
LANGUAGE: English
AB 1. The effects of various potential **inhibitors**, activators and inducers on the N-oxidn. of isomeric arom. diazines (pyrazine, **pyrimidine** and pyridazine) by rabbit liver microsomes have been studied. 2. 2,4-Dichloro-6-phenylphenoxyethylamine (DPEA), SKF 525-A and N-octylamine decreased N-oxide formation at 10-4M concns. 3. Methimazole and carbon monoxide **inhibited** the N-oxidn. of all three substrates studied. 4. The **inhibitory** effects were generally exaggerated when hepatic microsomal prepns. from phenobarbitone-pretreated animals were used as enzyme source. 5. When phenobarbitone or pyridine were used as inducing agents, the N-oxidn. of isomeric arom. diazines showed considerable induction, wheres .beta.-naphthoflavone and Aroclor 1254 pretreatment had much weaker effects. 6. It is suggested that P 4502E1 and/or 2B are the major subfamilies of P 450 involved in the N-oxidn. of isomeric diazines.

L9 ANSWER 13 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1995:689228 CAPLUS
DOCUMENT NUMBER: 123:340717
TITLE: Studies on the chemistry of **pyrimidine** derivatives with dimethyldioxirane: synthesis, cytotoxic effect and antiviral activity of new 5,6-oxiranyl-5,6-dihydro and 5-hydroxy-5,6-dihydro-6-substituted uracil derivatives and **pyrimidine** nucleosides
AUTHOR(S): Saladino, Raffaele; Bernini, Roberta; Crestini, Claudia; Mincione, Enrico; Bergamini, Alberto; Marini, Stefano; Palamara, Anna Teresa
CORPORATE SOURCE: Dip. Agrochim. Agrobiol., Univ. Viterbo "La Tuscia", Viterbo, 01100, Italy
SOURCE: Tetrahedron (1995), 51(27), 7561-78
CODEN: TETRAB; ISSN: 0040-4020
PUBLISHER: Pergamon
DOCUMENT TYPE: Journal
LANGUAGE: English
GI



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AB The oxidn. of uracil derivs. and **pyrimidine** nucleoside performed

in CH2Cl2 with dimethyldioxirane afforded new 5,6-oxiranyl-5,6-dihydro and cis-/trans-5,6-dihydroxy-5,6-dihydro-derivs. When the oxidns. were performed in the presence of methanol as nucleophile cis- and trans-5-hydroxy-6-methoxy-5,6-dihydro derivs. were obtained in acceptable yields. Cis- and trans-1,3-dimethyl-5-hydroxy-6-alkylamino-5,6-dihydro uracils were obtained by nucleophilic ring opening of the 1,3-dimethyl-5,6-oxiranyl-5,6-dihydro uracil in the purified form. Interestingly some of the new title products revealed low cytotoxicity and selective antiviral activity against DNA and RNA Viruses. In particular, compd. I shows a strong and selective **inhibition** of the Sendai virus with lower effect on Herpes Simplex-1 virus. Compd. I is also able to slightly **inhibit** HIV-1 virus at high concns., but in this case a cytotoxic effect was obsd.

L9 ANSWER 14 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1993:502416 CAPLUS
DOCUMENT NUMBER: 119:102416
TITLE: Quaternary polyamines as sulfite **oxidation inhibitors** in amine scrubbing of sulfur dioxide
INVENTOR(S): Bedell, Stephen A.
PATENT ASSIGNEE(S): Dow Chemical Co., USA
SOURCE: U.S., 12 pp. Cont.-in-part of U.S. 5,019,365.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 5167941	A	19921201	US 1990-623313	19901206
US 5019365	A	19910528	US 1990-546075	19900629

PRIORITY APPLN. INFO.: US 1988-277159 19881129
US 1990-546075 19900629
OTHER SOURCE(S): MARPAT 119:102416
AB SO32- oxidn. is **inhibited** in alk. scrubbing solns. for removal of SO2 from flue gases by adding 1-3000 ppm of a polyelectrolyte contg. quaternary ammonium groups (mol. wt. >10,000) to the scrubbing soln. The scrubbing soln. contains amines, e.g., piperazinones, morpholinones, piperidines, piperazines, piperazinediones, hydantoins, triazinones, pyrimidones, oxazolidones, and N-carboxymethyl ethylenediamines. Suitable polyelectrolytes include the reaction products of starch and chlorohydroxypropyl tri-Me ammonium salt or glycidyl tri-Me ammonium chloride, poly(diallyldimethylammonium chloride) and copolymers of acrylamide and quaternary ammonium compds.

L9 ANSWER 15 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1991:404696 CAPLUS
DOCUMENT NUMBER: 115:4696
TITLE: Method for making synthetic oligonucleotides which bind specifically to target sites on duplex DNA molecules, by forming a colinear triplex, the synthetic oligonucleotides and methods of use
INVENTOR(S): Hogan, Michael Edward; Kessler, Donald Joseph
PATENT ASSIGNEE(S): Baylor College of Medicine, USA
SOURCE: Eur. Pat. Appl., 40 pp.
CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 375408	A1	19900627	EP 1989-313391	19891220
EP 375408	B1	19950222		
R: GR				
CA 2006008	AA	19900620	CA 1989-2006008	19891219
WO 9006934	A1	19900628	WO 1989-US5769	19891220
W: AU, DK, JP				
RW: AT, BE, CH, DE, ES, FR, GB, IT, LU, NL, SE				
AU 9048384	A1	19900710	AU 1990-48384	19891220
AU 640910	B2	19930909		
EP 449972	A1	19911009	EP 1990-901460	19891220
R: AT, BE, CH, DE, ES, FR, GB, IT, LI, LU, NL, SE				
JP 04502407	T2	19920507	JP 1990-502252	19891220
ES 2069598	T3	19950516	ES 1989-313391	19891220
US 5176996	A	19930105	US 1989-453532	19891222
DK 9101200	A	19910620	DK 1991-1200	19910620
PRIORITY APPLN. INFO.:			US 1988-287359	19881220
			WO 1989-US5769	19891220

AB A method for making synthetic oligonucleotides which bind to target sequences in a duplex DNA forming colinear triplexes by binding to the major groove is disclosed. The method includes scanning genomic duplex DNA and identifying nucleotide target sequences .gtoreq.20 nucleotides having either .gtoreq.65% purine bases or .gtoreq.65% **pyrimidine** bases; and synthesizing synthetic oligonucleotides complementary to identified target sequences. The synthetic oligonucleotides have a G when the complementary location in the DNA duplex has a GC base pair and have a T when the complementary location in the DNA duplex has an AT base pair. The synthetic oligonucleotides are oriented 5' to 3' and bind parallel or 3' to 5' and bind antiparallel to the .gtoreq.65% purine strand. Also described are synthetic oligonucleotides made by the above methods. The oligonucleotides can be altered by modifying and/or changing the bases, adding linkers and modifying groups to the 5' and/or 3' termini, and changing the backbone. These synthetic oligonucleotides bind to duplex DNA to form triplexes. This process alters the functioning of the genes which are bound, and can be used to **inhibit** cell growth, alter protein ratios, treat diseases including cancer, and permanently alter the DNA. Oligonucleotides 3'-GTTTTTGGGTGTTGTGGGTGTGTGTGGTT-5' (HIV29par) and 5'-GTTTTTGGGTGTTGTGGGTGTGTGTGGTTTG-3' (HIV31 anti), designed to bind within the major groove of the DNA helix and form triplexes with specific sequences in the tar region of the human immunodeficiency virus 1 (HIV-1) provirus were readily taken up by HIV-1 infected cells and selectively suppressed synthesis of HIV-1 mRNA without concomitant suppression of mRNA for a constituent gene of the cells. **Inhibition** of viral mRNA was dependent on the dose of oligonucleotide added; max. **inhibition** occurred at 10 mM.

L9 ANSWER 16 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1991:159260 CAPLUS
DOCUMENT NUMBER: 114:159260
TITLE: Insertion of specific bases during DNA synthesis past the **oxidation**-damaged base 8-oxodG
AUTHOR(S): Shibutani, Shinya; Takeshita, Masaru; Grollman, Arthur P.
CORPORATE SOURCE: Dep. Pharm. Sci., State Univ. New York, Stony Brook, NY, 11794-8651, USA
SOURCE: Nature (London) (1991), 349(6308), 431-4
CODEN: NATUAS; ISSN: 0028-0836
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Oxidative damage to DNA, reflected in the formation of 8-oxo-7-hydrodeoxyguanosine (8-oxodG), may be important in mutagenesis, carcinogenesis and the ageing process. Y. Kuchino et al. (1987) studied

DNA synthesis on oligodeoxynucleotide templates contg. 8-oxodG, concluding that the modified base lacked base pairing specificity and directed misreading of **pyrimidine** residues neighboring the lesion. The present study reports different results, using an approach in which the several products of a DNA polymerase reaction can be measured. In contrast to the earlier report, it was found that dCMP and dAMP are incorporated selectively opposite 8-oxodG with transient **inhibition** of chain extension occurring 3' to the modified base. The potentially mutagenic insertion of dAMP is targeted exclusively to the site of the lesion. The ratio of dCMP to dAMP incorporated varies, depending on the DNA polymerase involved. Chain extension from the dA.cntdot.8-oxodG pair was efficiently catalyzed by all polymerases tested.

L9 ANSWER 17 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1989:511633 CAPLUS
DOCUMENT NUMBER: 111:111633
TITLE: Factors affecting the photooxidation of purine and **pyrimidine** bases sensitized by hypocrellin A
AUTHOR(S): Jia, Hongti; Dong, Cangyu; Wu, Yaonan
CORPORATE SOURCE: Dep. Biochem., Beijing Med. Univ., Beijing, Peop. Rep. China
SOURCE: Shengwu Huaxue Zazhi (1989), 5(3), 275-80
CODEN: SHZAE4; ISSN: 1000-8543
DOCUMENT TYPE: Journal
LANGUAGE: Chinese
AB Studies of hypocrellin A-sensitized photooxidn. of purine and **pyrimidine** bases were carried out using optical absorption and HPLC techniques. The UV absorbance of guanine and thymine in the presence of hypocrellin A (3 .times. 10-5M) at pH 9.0 decreased significantly after visible light irradn. for 40 min. A new peak occurred in the reversed-phase HPLC elution profile for guanine photooxidn. sensitized by hypocrellin A. The new peak had an absorption max. at 475 nm. It is proposed that the product(s) formed from hypocrellin A-sensitized photooxidn. of guanine might be cleavage product(s) of the purine ring. The rate of hypocrellin A-sensitized photooxidn. of guanine was dependent on pH, illumination time, and sensitizer concn. Azide, a specific quencher for 1O2, could partially **inhibit** the photooxidn. of guanine at a concn. of 40 mM and completely at concns. >110 mM. Thus, 1O2 is involved in hypocrellin A-sensitized photooxidn. of guanine. The possible photooxidn. pathway of guanine was discussed.

L9 ANSWER 18 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1989:226853 CAPLUS
DOCUMENT NUMBER: 110:226853
TITLE: **Inhibition** of autoxidation of divicine and isouramil by the combination of superoxide dismutase and reduced glutathione
AUTHOR(S): Winterbourn, Christine C.
CORPORATE SOURCE: Christchurch Sch. Med., Christchurch Hosp., Christchurch, N. Z.
SOURCE: Arch. Biochem. Biophys. (1989), 271(2), 447-55
CODEN: ABBIA4; ISSN: 0003-9861
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The effects of GSH on the autoxidn. of the fava bean **pyrimidine** aglycons, divicine and isouramil, and on acid-hydrolyzed vicine (provisional identification 2-amino-4,5,6-trihydroxypyrimidine) have been studied. GSH alone promoted redox cycling of each compd., with concomitant GSH oxidn. and H2O2 prodn. In the presence of superoxide dismutase, there is a lag period during which little **pyrimidine** oxidn. occurs, followed by a period of accelerated oxidn. With the three **pyrimidines**, increasing concns. of GSH extended this lag period

and progressively decreased subsequent rates of both **pyrimidine** oxidn. and O uptake. No GSH oxidn. or O uptake occurred during the lag. These results show that the combination of GSH and superoxide dismutase is able to **inhibit** redox cycling of the **pyrimidines**. With a 10-fold excess of GSH over isouramil or acid-hydrolyzed vicine (20-fold with divicine) this coupled oxidn. of GSH and the **pyrimidine** is almost completely suppressed. This mechanism may be a means whereby GSH in combination with superoxide dismutase protects against the cytotoxic effects of these reactive **pyrimidines**.

L9 ANSWER 19 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1989:149326 CAPLUS
DOCUMENT NUMBER: 110:149326
TITLE: Auto-**oxidation** of dialuric acid, divicine and isouramil. Superoxide dependent and independent mechanisms
AUTHOR(S): Winterbourn, Christine C.; Cowden, William B.; Sutton, Harry C.
CORPORATE SOURCE: Christchurch Sch. Med., Christchurch Hosp., Christchurch, N. Z.
SOURCE: Biochem. Pharmacol. (1989), 38(4), 611-18
CODEN: BCPCA6; ISSN: 0006-2952
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The toxicity of dialuric acid to pancreatic .beta. cells, and the hemolytic action of divicine and isouramil involve auto-oxidn. and redox cycling reactions. Divicine and isouramil are produced on hydrolysis of the fava bean glycosides, vicine and convicine. The mechanism of auto-oxidn. of the 3 compds. as well as the acid hydrolysis product of vicine (provisionally assigned the structure 2-amino-4,5,6-trihydroxypyrimidine) has been studied. All 4 **pyrimidines** auto-oxidized rapidly at neutral pH, generating H2O2 by an O-dependent chain mechanism. Superoxide dismutase-**inhibited** lag period varied with pH, temp., and **pyrimidine** concn., and was much shorter for isouramil and divicine than for dialuric acid and acid-hydrolyzed vicine. The initial rate of dialuric acid oxidn. was greater and the acceleration less pronounced than with the other **pyrimidines**. A mechanism common to all 4 **pyrimidines** has been shown by kinetic anal. to account for nearly all the observations in the presence and absence of superoxide dismutase. Autocatalysis in the latter case is attributed mainly to the reactions reduced **pyrimidine** + oxidized **pyrimidine** .dblharw. 2 **pyrimidine** radical and **pyrimidine** radical + O2 .fwdarw. oxidized **pyrimidine** + O2-. Rate consts. for these and other reactions are reported. At pH 7.4 and 37.degree., the lag period before 100 .mu.M acid-hydrolyzed vicine underwent rapid oxidn. was .apprx.15 min. Isouramil and divicine at an equiv. concn. gave lags of <1 min, which became less at higher concns. Thus, intracellular superoxide dismutase should provide only transitory protection against the oxidn. products of dialuric acid, divicine, or isouramil. Prolonged protection should only be achieved if the accumulation of oxidized **pyrimidine** is also prevented.

L9 ANSWER 20 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1987:593866 CAPLUS
DOCUMENT NUMBER: 107:193866
TITLE: The **oxidation** of 4-**pyrimidinone** and 4-quinazolinone and their N-methyl derivatives by milk xanthine oxidase
AUTHOR(S): Bunting, John W.; Luscher, Mark A.; Redman, Jane
CORPORATE SOURCE: Dep. Chem., Univ. Toronto, Toronto, ON, M5S 1A1, Can.
SOURCE: Bioorg. Chem. (1987), 15(2), 125-40
CODEN: BOCMBM; ISSN: 0045-2068

DOCUMENT TYPE: Journal
LANGUAGE: English
OTHER SOURCE(S): CASREACT 107:193866

AB 4-**Pyrimidinone**, 4-quinazolinone, and each of their N1-Me derivs. are oxidized to the corresponding 2,4-diones by milk xanthine oxidase. Steady-state kinetic parameters were evaluated for the enzymic oxidn. of these substrates over the pH range 5.0-10.5. The pH dependences of kcat/Km (kcat = catalytic const.) for 4-**pyrimidinone** and 4-quinazolinone are consistent with the neutral mols. of these species being substrates, but their anionic conjugate bases not being enzymically oxidized. Apart from this substrate ionization, kcat and Km do not show any dramatic pH dependence. 1-Ethyl-4-**pyrimidinone** is slowly oxidized by this enzyme, and 3-methyl-4-**pyrimidinone** is an extremely poor substrate; 3-methyl-4-quinazolinone is not enzymically oxidized. These latter 2 species are competitive **inhibitors** for the oxidn. of 4-**pyrimidinone**. The 2- and 4-**pyrimidinones**, the 2- and 4-quinolinones, 1-isoquinolinone, and each of their N-Me derivs. were reversible **inhibitors** for this enzyme and ID50 values (concns. giving half-maximal **inhibition**) values were evaluated. These data are consistent with the neutral 1H tautomers of 4-**pyrimidinone** and 4-quinazolinone being the true substrates for this enzyme. The low reactivity of 4-quinazolinone as a substrate can probably be traced to reversible **inhibition** by 4(3H)-quinazolinone of the enzymic oxidn. of 4(1H)-quinazolinone.

L9 ANSWER 21 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1985:199779 CAPLUS
DOCUMENT NUMBER: 102:199779
TITLE: Site-specific DNA damage caused by lipid peroxidation products
AUTHOR(S): Ueda, Kazumitsu; Kobayashi, So; Morita, Junji; Komano, Tohru
CORPORATE SOURCE: Dep. Agric. Chem., Kyoto Univ., Kyoto, 606, Japan
SOURCE: Biochim. Biophys. Acta (1985), 824(4), 341-8
CODEN: BBACAQ; ISSN: 0006-3002
DOCUMENT TYPE: Journal
LANGUAGE: English

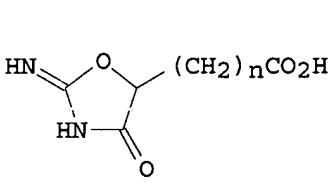
AB DNA damage induced by autoxidized lipids was investigated using covalently closed, circular (supercoiled) DNA and DNA fragments of defined sequence. DNA strand-breaking substances accumulated during autoxidn. of Me linolenate, and strand breakage was measured with samples taken at different times. The DNA strand-breaking activity reached its max. a little after the peak value of peroxide and decreased upon further autoxidn. The peak of the DNA strand-breaking activity did not always coincide with the peak of thiobarbituric acid reactants or of conjugated diene, either. The DNA strand-breaking reaction was dependent on metal ions and was **inhibited** by KI and tiron and partially by catalase, suggesting the involvement of radical species and(or) O radicals. No direct cleavage of singly end-labeled 100-200 base-pair DNA fragments by autoxidized Me linolenate and Cu2+ was detected under the conditions used. Cleavage occurred during subsequent heating in piperidine after the reaction. The alkali-labile damage was preferentially induced at **pyrimidine** residues, esp. in dinucleotide sequences of **pyrimidine**-guanine (5'.fwdarw.3'), as detd. by sequencing.

L9 ANSWER 22 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1985:45928 CAPLUS
DOCUMENT NUMBER: 102:45928
TITLE: 1,3-Oxazolidin-4-one derivatives
INVENTOR(S): Krepelka, Jiri; Pouzar, Vladimir
PATENT ASSIGNEE(S): Czech.
SOURCE: Czech., 2 pp.

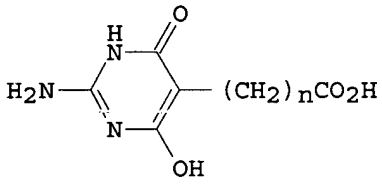
CODEN: CZXXA9
DOCUMENT TYPE: Patent
LANGUAGE: Czech
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
CS 206095	B	19810630	CS 1979-5318	19790801

GI



I



II

AB Five oxazolidinone derivs. I (n = 1-5) were prepd. by oxidn. of **pyrimidines** II with aq. H2O2 or air. I had antineoplastic and anticonvulsant activity in exptl. animals (no data).

L9 ANSWER 23 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1984:579922 CAPLUS
DOCUMENT NUMBER: 101:179922
TITLE: Electrochemical process for the preparation of sulfoxides of thioformamide derivatives, useful as medicaments [for treating hypertension]
INVENTOR(S): Bizot, Jean; Deprez, Dominique
PATENT ASSIGNEE(S): Rhone-Poulenc Sante, Fr.
SOURCE: S. African, 16 pp.
CODEN: SFXXAB
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
ZA 8304438	A	19840328	ZA 1983-4438	19830616
DD 210082	A5	19840530	DD 1983-252090	19830616
US 4466866	A	19840821	US 1983-504788	19830616
AT 23577	E	19861115	AT 1983-401243	19830616
PRIORITY APPLN. INFO.:			DD 1983-252090	19830616
			EP 1983-401243	19830616
			ZA 1983-4438	19830616

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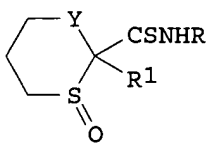


I

AB The sulfoxides I (R = H or a 1-4 C alkyl group, R' = heterocyclic group of arom. character contg. 1 or 2 N atoms selected from pyridin-3-yl (optionally substituted by a 1-4 C (or halogen) contg. group), quinolin-3-yl, pyridazin-4-yl, **pyrimidin**-5-yl, thiazol-5-yl, thieno[2,3-b]pyridin-5-yl and thieno[3,2-b]pyridin-6-yl, and Y is a valency bond or methylene group) are obtained by the electrochem. oxidn. of the ring S atom of a thioformamide deriv. N-Methyl-2-(pyridin-3-yl)tetrahydrothiophen-2-carbothioamide [82081-31-0] is electrochem. oxidized to form trans-N-methyl-2-(pyridin-3-yl)tetrahydrothiophen-2-carbothioamide 1-oxide [92569-64-7].

L9 ANSWER 24 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1983:422835 CAPLUS
DOCUMENT NUMBER: 99:22835
TITLE: Pattern of hydroxyl radical addition to cytosine and 1-, 3-, 5-, and 6-substituted cytosines. Electron transfer and dehydration reactions of the hydroxyl adducts
AUTHOR(S): Hazra, D. K.; Steenken, S.
CORPORATE SOURCE: Max-Planck-Inst. Strahlenchem., Muelheim, D-4330, Fed. Rep. Ger.
SOURCE: J. Am. Chem. Soc. (1983), 105(13), 4380-6
CODEN: JACSAT; ISSN: 0002-7863
DOCUMENT TYPE: Journal
LANGUAGE: English

AB By use of the technique of pulse radiolysis with optical detection, the isomer distribution of the radicals formed in aq. soln. by addn. of OH radicals to cytosine and its derivs., e.g., 5-methylcytosine, 3-methylcytosine, cytidine, 5-methylcytidine, cytidylic acid and 2'-deoxycytidine, has been detd. by utilizing differences between the isomeric OH adducts with resp. to electron-transfer reactions with the reductant N,N,N',N'-tetramethyl-p-phenylenediamine (TMPD) or the oxidant tetranitromethane (TNM). The radicals of Cy-5-OH, formed by addn. of OH to C(5) of cytosine (87% of the OH radicals), 3- and 5-methylcytosine (92% and 65%, resp.), 5-carboxycytosine (82%), and 2-amino-4-hydroxy-6-methylpyrimidine (95%) reduce TNM to yield nitroform anion. The radicals Cy-6-OH, formed by addn to C(6), oxidize TMPD to TMPS+. The Cy-5-OH radicals undergo a base-catalyzed dehydration to yield readicals that are able to oxidize TMPD to TMPD+. In the case of cytosine the dehydrated OH adduct is identical with the one-electron oxidn. product from the reaction of cytosine with SO4-.cntdot.. If N(1) of the **pyrimidine** ring is substituted as with 1-methylcytosine, cytidine, cytidylic acid, 2'-deoxycytidine and 2'-deoxycytidylic acid, no dehydration of the OH adducts occur. In contrast, substitution by alkyl at N(3) does not **inhibit** the dehydration of the corresponding Cy-5-OH radical. In the presence of oxygen the Cy-5-OH and Cy-6-OH radicals are converted into peroxyl radicals which oxidize TMPD. In basic soln. these peroxyl radicals decomp., presumably by elimination of O2-.cntdot.. With 5-methylcytosine a peroxyl radical derived from the radical formed by H abstraction from the Me group is addnl. obsd. With the cytosine nucleosides and nucleotides the probability of OH attack at the cytosine mol. is >80%.

L9 ANSWER 25 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1982:178179 CAPLUS
DOCUMENT NUMBER: 96:178179
TITLE: Comparative effects of ancymidol and its analogs on growth of peas and ent-kaurene **oxidation** in cell-free extracts of immature Marah macrocarpus endosperm
AUTHOR(S): Coolbaugh, Ronald C.; Swanson, David I.; West, Charles A.
CORPORATE SOURCE: Dep. Botany, Iowa State Univ., Ames, IA, 50011, USA

SOURCE: Plant Physiol. (1982), 69(3), 707-11
CODEN: PLPHAY; ISSN: 0032-0889
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The plant growth retardant .alpha.-cyclopropyl-.alpha.-(4-methoxyphenyl)-5-**pyrimidine** Me alc. (ancymidol) and a series of its analogs having one or more of the substituents varied, were tested for their comparative biol. activity. The compds. were tested as **inhibitors** of internode elongation in peas and as **inhibitors** of the oxidn. of ent-kaurene catalyzed by microsomal prepns. from the liq. endosperm of M. macrocarpus seeds. The relative effectiveness of a substance was generally the same as an **inhibitor** of the 2 processes. Ancymidol was the most effective. Substitution of the alc. group of ancymidol by either Me or H groups reduced the activity only slightly. Substitution of the cyclopropyl group by an iso-Pr moiety also had little effect on the activity. However, substitution of the cyclopropyl group with a Ph or other aryl substituent greatly reduced the effectiveness of the analog as an **inhibitor**. Replacement of the 4-methoxyphenyl substituent with a similar substituent such as 4-chlorophenyl had little effect on activity, but replacement with a 2-methoxyphenyl group greatly reduced activity. Analogs in which the pyrimidyl moiety of ancymidol was modified were inactive in whole plants, but moderately active in the cell-free ent-kaurene oxidn. system. The application of gibberellic acid can overcome the growth **inhibitions** caused by treatment of the test plants with .ltoreq.10-5M of the **inhibitors**. However, the **inhibitory** effects of .gtoreq.10-4M **inhibitors** on test plants were not overcome by the applications of exogenous gibberellic acid. The effects of low concns. of these substances on plant growth are primarily a consequence of **inhibition** of ent-kaurene oxidn. and gibberellin biosynthesis. Other modes of **inhibition** may operate at higher **inhibitor** concns.

L9 ANSWER 26 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1982:47794 CAPLUS
DOCUMENT NUMBER: 96:47794
TITLE: **Oxidation** of **pyrimidine** and purine deoxyribonucleosides by reactive oxygen species generated by the hydrolytic decomposition of potassium perchromate
AUTHOR(S): Cadet, J.; Balland, A.; Voituriez, L.; Hahn, B. S.; Wang, S. Y.
CORPORATE SOURCE: Lab. Radiobiochim., CEN, Grenoble, Fr.
SOURCE: Oxygen Oxy-Radicals Chem. Biol., [Proc. Int. Conf.] (1981), Meeting Date 1980, 610-11. Editor(s): Rodgers, Michael A. J.; Powers, Edward Lawrence. Academic: New York, N. Y.
CODEN: 46WOAA
DOCUMENT TYPE: Conference
LANGUAGE: English

AB Anal. of the decompn. products resulting from K3CrO8 oxidn. of deoxyribothymidine, deoxyribocytosine, deoxyriboadenosine, and deoxyriboguanosine suggested that **pyrimidine** oxidn., which is **inhibited** by the OH.bul. scavenger MeOH, results from OH.bul. addn. to the 5,6-**pyrimidine** bond followed by the fast reaction of O2, whereas purine oxidn., which is **inhibited** by the singlet O scavenger N3, is due to singlet O generation. O2- which is also produced in the K3CrO8 reaction is not reactive toward the nucleosides. Thus, K3CrO8 is a convenient oxidizing agent for the selective degrdn. of DNA components either by OH.bul. or single O.

L9 ANSWER 27 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1979:518847 CAPLUS
DOCUMENT NUMBER: 91:118847

TITLE: Catalytic release of deoxyribonucleic acid bases by **oxidation** and reduction of an iron .cntdot. bleomycin complex
AUTHOR(S): Povirk, Lawrence F.
CORPORATE SOURCE: Dep. Mol. Biophys. Biochem., Yale Univ., New Haven, CT, 06520, USA
SOURCE: Biochemistry (1979), 18(18), 3989-95
CODEN: BICHAW; ISSN: 0006-2960
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The kinetics and stoichiometry of several reactions involving bleomycin, Fe, DNA, O, and sulfhydryls were examd. in order to assess their possible role in degrdn. of DNA by bleomycin. Oxidn. of Fe(II) in the presence of bleomycin resulted in an Fe(III).cntdot.bleomycin complex, having an optical absorption spectrum with a broad shoulder at 320-400 nm, which was stable for several hours. If Fe(II) was allowed to oxidize before bleomycin addn., the complex did not form. The complex was reduced by dithiothreitol 5 times faster than unchelated Fe(III), and redn. of the complex was **inhibited** by high concns. of DNA. However, stopped-flow studies showed that, when sufficient DNA was present to bind most of the Fe(II).cntdot.bleomycin, its rate of oxidn. by O was 60 times faster than that of unbound Fe(II).cntdot.bleomycin. Under the same conditions, oxidn. of each mol of DNA-bound Fe(II).cntdot.bleomycin released 0.18 mol of thymine. Treatment of **pyrimidine**-labeled Escherichia coli DNA with bleomycin and high concns. of Fe(II) and 2-mercaptoethanol resulted in the release of .ltoreq.2.4 mol of **pyrimidines** (of which 60% were thymine) per mol of bleomycin. This result implies that each Fe.cntdot.bleomycin complex went through several cycles of oxidn. and redn. and that bleomycin usually was not inactivated in the base-release reaction. In supercoiled plasmid pDR3709 DNA, 1 base was released per single-strand break (measured in alkali), eliminating the possibility of multiple base release during a single bleomycin-DNA interaction. Thus, the Fe.cntdot.bleomycin complex acts as a catalyst which, after being reduced by sulfhydryls, binds to DNA in a way which facilitates both the oxidn. of the chelated Fe(II) and the degrdn. of the DNA backbone by the products of this oxidn.

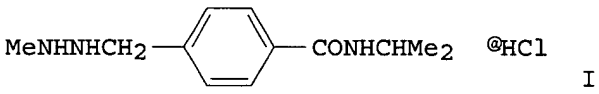
L9 ANSWER 28 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1977:596313 CAPLUS
DOCUMENT NUMBER: 87:196313
TITLE: **Oxidation** of hypoxanthines, bearing 8-aryl or 8-pyridyl substituents, by bovine milk xanthine oxidase
AUTHOR(S): Bergmann, Felix; Levene, Lawrence; Govrin, Hanna
CORPORATE SOURCE: Hadassah Med. Sch., Hebrew Univ., Jerusalem, Israel
SOURCE: Biochim. Biophys. Acta (1977), 484(2), 275-89
CODEN: BBACAQ
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Hypoxanthines, contg. aryl or pyridyl substituents at position 8, were converted by bovine milk xanthine oxidase into their corresponding xanthines at low rates. Oxidn. was accelerated considerably when the 8-pyridyl substituents were quaternized. In the enzymic oxidn. of quaternary 8-pyridylhypoxanthines, a lag phase preceded the attainment of a const., max. reaction rate. The delay is assumed to be due to a relatively slow conformational change in the active enzymic center. In 8-(3'-N-methylpyridinio)xanthine betaine, the pyridinium moiety was also attacked at high pH (9-11) to yield an N-methyl-2-pyridone. The analogous pyridone was the only oxidn. product of 1-methyl-8-(3'-N-methylpyridinio)hypoxanthine betaine, which was not attacked in the **pyrimidine** ring. The cationic substrates were attracted to the enzyme by an anionic group, which probably formed an ion pair with a protonated NH2 group in or near the active center.

L9 ANSWER 29 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1977:67403 CAPLUS
DOCUMENT NUMBER: 86:67403
TITLE: Influence of 8-substituents on the **oxidation** of hypoxanthine and 6-thioxopurine by bovine milk xanthine oxidase
AUTHOR(S): Bergmann, Felix; Levene, Lawrence; Govrin, Hanna; Frank, Aryeh
CORPORATE SOURCE: Hadassah Med. Sch., Hebrew Univ., Jerusalem, Israel
SOURCE: Biochim. Biophys. Acta (1977), 480(1), 39-46
CODEN: BBACAQ
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The effect of 8-substituents on the rate of oxidn. of hypoxanthine and 6-thioxopurine by bovine milk xanthine oxidase was studied. An 8-Me group does not alter the rate of oxidn. of hypoxanthine materially, but an 8-Ph substituent reduces it markedly. This is ascribed to **inhibition** of the tautomerization process, responsible for substrate activation, prior to oxidn. In contrast, the 8-Ph group in 3-methyl-8-phenylhypoxanthine enhances the rate, presumably by binding to a hydrophobic site near the enzymic center. An 8-Ph group in 6-thioxopurine markedly increases the rate of enzymic oxidn. Probably the arom. substituent diverts anion formation to the imidazole ring. In contrast, ionization of 8-methyl-6-thioxopurine involves the **pyrimidine** moiety, thus rendering enzymic attack at position 2 more difficult.

L9 ANSWER 30 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1977:66977 CAPLUS
DOCUMENT NUMBER: 86:66977
TITLE: Kinetic study of the DNA-natulan reaction mechanism. Part III. NMR investigation of the effect of nucleotides on natulan **oxidation**
AUTHOR(S): Belova, L. A.; Zenin, S. V.; Emanuel, N. M.
CORPORATE SOURCE: Chem. Fac., Moscow State Univ., Moscow, USSR
SOURCE: Stud. Biophys. (1976), 60(3), 171-9
CODEN: STBIBN
DOCUMENT TYPE: Journal
LANGUAGE: Russian
GI

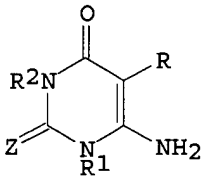


I

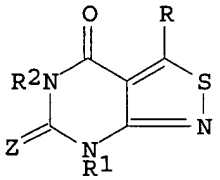
AB The autoxidn. of natulan (I) in the presence and absence of ribonucleoside monophosphates was analyzed by 1H NMR in phosphate buffer in D2O. During autoxidn. in the absence of nucleotides, the intensities of the resonance lines for the NMe, Me2CH, and CH2 groups of I decreased, and that for the C6H6 ring H atoms disappeared and was replaced by a broad unresolved band. Two new resonance lines also appeared that were assigned to NMe groups of reaction products. Thus, autoxidn. of I may initially consist of the cleavage of its CH2-NH band. Similar changes were obsd. during the autoxidn. of I in the presence of nucleotides. However, the rate of autoxidn. of 0.1M I was significantly decreased by 0.2M AMP; moderately decreased by 0.2M CMP, 0.1M rTMP, and(or) 0.2M UMP; and slightly decreased by 0.02M GMP. At equiv. concns., GMP and AMP may exhibit similar **inhibitory** effects. The purine nucleotides may exhibit higher **inhibitory** activities than the **pyrimidine** nucleotides as the result of the increased ability of the former to form complexes with

I. The presence of nucleotides stabilized a reaction product that was identified as methylhydrazine. Thus, I can undergo autoxidn. in nucleotide complexes and, possibly, also in DNA complexes. The subsequent hydrolysis of the hydrazine may cause the cleavage of the DNA chain which was previously obsd. in DNA-natulan solns.

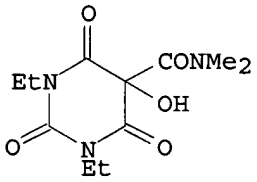
L9 ANSWER 31 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1976:560012 CAPLUS
DOCUMENT NUMBER: 85:160012
TITLE: Fused **pyrimidines**. II. Synthesis and **oxidation** of 3-aminoisothiazolo[3,4-d] **pyrimidines**
AUTHOR(S): Furukawa, Yoshiyasu; Shima, Shunsuke
CORPORATE SOURCE: Cent. Res. Div., Takeda Chem. Ind., Ltd., Osaka, Japan
SOURCE: Chem. Pharm. Bull. (1976), 24(5), 979-86
CODEN: CPBTAL
DOCUMENT TYPE: Journal
LANGUAGE: English
GI



I, R=H, Z=O,S
II, R=C(S)NHR3, Z=O,S



III, R=NHR3
IV, R=NR3R4



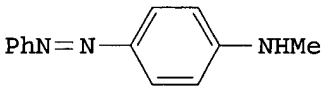
V

AB The 6-aminouracils I (R1 = PhCH2, Et, H, Me, p-ClC6H4, MeOCH2CH2, Ph, Me2CHCH2, p-MeOC6H4; R2 = H, Et) reacted with SCNR3 (R3 = Et, Me, C6H4Cl-p, Ph, C6H4OH-p, CH2Ph) to give the thiocarbamoyluracils II, which were oxidized with Br or H2O2 to give the isothiazolopyrimidinediones III, whose alkylation with R4I (R4 = Me, Et) gave the (disubstituted amino)isothiazolopyrimidines IV. Further oxidn. of III or IV (R1 = R2 = Et, R3 = R4 = Me) with H2O2 gave the hydroxybarbituric acid V. Aminoisothiazolopyrimidines are potential cyclic nucleotide phosphodiesterase **inhibitors**.

L9 ANSWER 32 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1976:518637 CAPLUS
DOCUMENT NUMBER: 85:118637
TITLE: **Oxidation** of selected pteridine derivatives by mammalian liver xanthine oxidase and aldehyde oxidase
AUTHOR(S): Hodnett, C. N.; McCormack, J. J.; Sabean, J. A.
CORPORATE SOURCE: Coll. Med., Univ. Vermont, Burlington, Vt., USA
SOURCE: J. Pharm. Sci. (1976), 65(8), 1150-4
CODEN: JPMSAE
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Xanthine oxidase, obtained from rat liver, oxidizes a variety of substituted amino- and hydroxypteridines in a manner identical to that previously obsd. for milk xanthine oxidase. For example, 2-aminopteridine and its 4- and 7-OH derivs. are oxidized efficiently to 2-amino-4,7-dihydropteridine (isoxanthopterin) by the rat liver enzyme and 4-aminopteridine and its 2- and 7-OH derivs. are oxidized to 4-amino-2,7-dihydroxypteridine. 4-Hydroxypteridine and the isomeric 2-

and 7-hydroxypteridines are oxidized by rat liver xanthine oxidase to 2,4,7-trihydroxypteridine. Rabbit liver aldehyde oxidase, but not rat liver xanthine oxidase, catalyzes the oxidn. in position 7 of 2,4-diaminopteridine and its 6-Me and 6-hydroxymethyl derivs. 2-Aminopteridine and 4-aminopteridine are both oxidized to the corresponding 7-OH derivs. in the aldehyde oxidase system; 2-amino-4-hydroxypteridine appears to be a minor product in the **oxidation** of 2-aminopteridine by rabbit liver aldehyde oxidase. Both aldehyde oxidase and xanthine oxidase catalyze the oxidn. of 2-amino-6,7-disubstituted pteridines to the corresponding 4-OH derivs.; 4-hydroxy-6,7-disubstituted pteridines are oxidized in position 2 by both enzymes. 4-Amino-6,7-disubstituted pteridines are not oxidized by either enzyme. 2-Amino-4-methylpteridine is oxidized in position 7 by aldehyde oxidase but is not an effective substrate for xanthine oxidase; 2-hydroxypteridine and 7-hydroxypteridine are not oxidized to a detectable extent by aldehyde oxidase. All oxidns. mediated by xanthine oxidase are strongly **inhibited** by allopurinol (4-hydroxypyrazolo[3,4-d] **pyrimidine**), and all oxidns. medited by aldehyde oxidase are **inhibited** by menadione (2-methyl-1,4-naphthoquinone). Rat liver xanthine oxidase and, to a lesser extent, rabbit liver aldehyde oxidase are **inhibited** by 4-chloro-6,7-dimethylpteridine; 2-amino-3-pyrazinecarboxylic acid **inhibits** xanthine oxidase but not aldehyde oxidase. The oxidns. of 2- and 4-aminopteridines by aldehyde oxidase results in concomitant redn. of cytochrome c.

L9 ANSWER 33 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1976:174883 CAPLUS
DOCUMENT NUMBER: 84:174883
TITLE: Microsomal N-**oxidation** of the hepatocarcinogen N-methyl-4-aminoazobenzene and the reactivity of N-hydroxy-N-methyl-4-aminoazobenzene
AUTHOR(S): Kadlubar, Fred F.; Miller, James A.; Miller, Elizabeth C.
CORPORATE SOURCE: Med. Sch., Univ. Wisconsin, Madison, Wis., USA
SOURCE: Cancer Res. (1976), 36(3), 1196-206
CODEN: CNREA8
DOCUMENT TYPE: Journal
LANGUAGE: English
GI

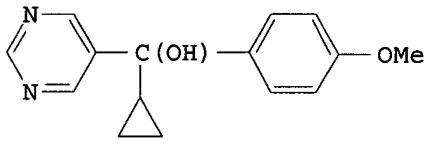


I

AB The N-oxidn. of N-methyl-4-aminoazobenzene (MAB)(I) [621-90-9] was catalyzed by hepatic microsomes in a reduced pyridine nucleotide- and oxygen-dependent reaction. The initial N-oxidn. product, N-hydroxy-N-methyl-4-aminoazobenzene (N-HO-MAB) [1910-36-7], was readily oxidized to a second product that yielded N-hydroxy-4-aminoazobenzene [6530-27-4] upon subsequent acid treatment. The secondary N-oxidn. product may be formed nonenzymatically and is presumed to be N-HO-MAB N-oxide [58989-03-0] or its dehydrated deriv., N-(p-phenylazophenyl)nitrone [58989-01-8]. Under the same conditions, MAB was also oxidatively N-dealkylated to 4-aminoazobenzene [60-09-3], which was N-oxidized to N-hydroxy-4-aminoazobenzene [6530-27-4]. Unlike the latter reactions, the microsomal N-oxidn. of MAB was independent of cytochrome P-450, as shown by its lack of sensitivity to **inhibition** by 2-[(2,4-dichloro-6-phenyl)phenoxy]ethylamine and its inability to utilize cumene hydroperoxide in place of reduced pyridine nucleotides and oxygen. The N-oxidn. of MAB was also catalyzed by the purified microsomal

flavoprotein mixed-function amine oxidase [9059-11-4]. The noncarcinogenic dye N-ethyl-4-aminoazobenzene [2058-67-5] was metabolized similarly to MAB. For male animals the hepatic levels of MAB N-oxidase [59088-29-8] activity were in the order: rat > hamster, guinea pig > mouse, rabbit. Little or no MAB N-oxidase activity was present in several extrahepatic rat tissues. N-HO-MAB, N-hydroxy-N-ethyl-4-aminoazobenzene [58989-02-9], and N-hydroxy-4-aminoazobenzene catalyzed the aerobic oxidn. of cysteine and glutathione. These hydroxylamines also bound covalently to proteins. The binding of N-HO-MAB with nucleic acids was only 3 to 6% that obsd. with serum albumin. Under anhydrous conditions the nitrone generated aerobically from N-HO-MAB reacted with carbon-carbon or carbon-nitrogen double bonds, or both, in fatty acids, retinol, purines, and **pyrimidines** to yield isoxazolidine and/or oxadiazolidine addn. products. The nitrone from N-hydroxy-N-ethyl-4-aminoazobenzene was much less reactive under these conditions. Syntheses of N-HO-MAB and N-hydroxy-N-ethyl-4-aminoazobenzene are reported.

L9 ANSWER 34 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1976:145955 CAPLUS
DOCUMENT NUMBER: 84:145955
TITLE: **Inhibition** of ent-kaurene **oxidation** and growth by .alpha.-cyclopropyl-.alpha.-(p-methoxyphenyl)-5-**pyrimidine** methyl alcohol
AUTHOR(S): Coolbaugh, Ronald C.; Hamilton, Roxanne
CORPORATE SOURCE: Dep. Nat. Sci. Math., Oregon Coll. Educ., Monmouth, Oreg., USA
SOURCE: Plant Physiol. (1976), 57(2), 245-8
CODEN: PLPHAY
DOCUMENT TYPE: Journal
LANGUAGE: English
GI



I

AB Growth of Alaska peas (Pisum sativum) was **inhibited** >60% by 19.5 and 39 .mu.M ancymidol (I) [12771-68-5] treatment. This growth **inhibition** was reversed completely by gibberellic acid [77-06-5] application. Cell-free enzyme prepns. from pea shoot tips and wild cucumber (Marah oregana) endosperm were used to test the effects of this substituted **pyrimidine** on the incorporation of mevalonic acid-14C into ent-kaurene [562-28-7] and ent-kaurenol [2300-11-0], resp. I (10-6M) completely blocked the conversion of ent-kaurene to ent-kaurenol. I at higher concns. (10-3M) **inhibited** the incorporation of mevalonic acid-14C into ent-kaurene less than it did at lower I concns. One mode of action of I is the **inhibition** of gibberellin biosynthesis.

L9 ANSWER 35 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1976:74294 CAPLUS
DOCUMENT NUMBER: 84:74294
TITLE: Pyrido[2,3-d]**pyrimidinedione** derivatives
INVENTOR(S): Noda, Kanji; Nakagawa, Akira; Miyata, Satoru; Motomura, Toshiharu; Ide, Hiroyuki
PATENT ASSIGNEE(S): Hisamitsu Pharmaceutical Co., Inc., Japan
SOURCE: Japan. Kokai, 6 pp.
CODEN: JKXXAF

09/ 891,671

DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 50100090	A2	19750808	JP 1974-5049	19740105
JP 57061757	B4	19821225		

GI For diagram(s), see printed CA Issue.

AB Pyrido[2,3-d]**pyrimidinediones** I (R1 = aryl, cycloalkyl, aralkyl; R2 = substituted alkyl, unsatd. alkyl) were prepd. by oxidn. of II (Z = CO, CS, CH2; X = halo, inorg. or org. ester group). I had analgesic, antiinflammatory, and central nerve depressant activities (no data). Thus, a mixt. of 3 g II (R1 = m-F3CC6H4, R2 = Et, Z = CH2, X = EtSO4), 5 g K2Cr2O7, and 100 ml concd. H2SO4-H2O (1:1) was refluxed 3 hr to give 1.8 g I (R1 = m-F3CC6H4, R2 = Et). Among 152 more I prepd. were (R1, R2 given): m-BrC6H4, Me; Ph, Me; Ph, Et; and Ph, Pr.

L9 ANSWER 36 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1975:589717 CAPLUS
DOCUMENT NUMBER: 83:189717
TITLE: **Oxidation** of 7-aminothiadiazolo(3,4-d) **pyrimidines** and 7-aminofurazano(3,4-d) **pyrimidines** by xanthine oxidase and aldehyde oxidase
AUTHOR(S): McCormack, John J.; Taylor, Edward C.
CORPORATE SOURCE: Coll. Med., Univ. Vermont, Burlington, Vt., USA
SOURCE: Biochem. Pharmacol. (1975), 24(17), 1636-9
CODEN: BCPCA6
DOCUMENT TYPE: Journal
LANGUAGE: English

GI For diagram(s), see printed CA Issue.

AB Oxidn. of 7-aminothiadiazolo[3,4-d]**pyrimidine** (I) by xanthine oxidase from milk (Km 5 .times. 10-5M) occurred at the unsubstituted 5 position of the heterocyclic ring, yielding 7-amino-5-hydroxythiadiazolo[3,4-d]**pyrimidine**. Oxidn. of I by aldehyde oxidase was similar to that by xanthine oxidase, with a Km of 1.2 .times. 10-4M. Oxidn. of 7-aminofurazano[3,4-d]**pyrimidine** (II) by xanthine oxidase occurred with a Km of 7 .times. 10-5M, and that by aldehyde oxidase with a Km 1.5 .times. 10-4M. The 5-methyl, 5-amino, and 5-methylthio derivs. of II acted neither as substrates nor **inhibitors** of the enzymes.

L9 ANSWER 37 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1973:463815 CAPLUS
DOCUMENT NUMBER: 79:63815
TITLE: Distribution, subcellular localization, and product **inhibition** of dihydroorotate **oxidation** in the rat
AUTHOR(S): Kennedy, James
CORPORATE SOURCE: Coll. Physicians Surg., Columbia Univ., New York, N. Y., USA
SOURCE: Arch. Biochem. Biophys. (1973), 157(2), 369-73
CODEN: ABBIA4
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The enzyme system for converting dihydroorotate to orotate was distributed in all rat tissues assayed. In liver, the reaction is localized in mitochondria and appears to be specific for dihydroorotate. Evidence is presented for excluding the reaction as a site for control of **pyrimidine** biosynthesis via end-product **inhibition**. However, product **inhibition** by orotic acid does occur and is

competitive with dihydroorotate.

L9 ANSWER 38 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1973:144682 CAPLUS
DOCUMENT NUMBER: 78:144682
TITLE: **Inhibition** of ferritin reduction by pyrazolo[3,4d]**pyrimidines**
AUTHOR(S): Duggan, D. E.; Streeter, K. B.
CORPORATE SOURCE: Merck Inst. Ther. Res., West Point, Pa., USA
SOURCE: Arch. Biochem. Biophys. (1973), 156(1), 66-70
CODEN: ABBIA4
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The characteristics of **inhibition** of the ferritin reductase function of xanthine oxidase by 3 pyrazolopyrimidines is described. Under anaerobic conditions, the ferritin reduction coupled to hypoxanthi **oxidation** was **inhibited** by the 3 pyrazolopyrimidines tested, 4- hydroxyprazoloprimidine, 4,6-dihydroxyprazoloprimidine, and 4-mercaptopyrazolopyrimidine. Under the same conditions, 4 purine analogs were devoid of **inhibitory** activity, and 8-azaguanine and 8-azaadenine were weakly **inhibitory**.

L9 ANSWER 39 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1973:83315 CAPLUS
DOCUMENT NUMBER: 78:83315
TITLE: Sulfanilamides as **inhibitors** of **oxidation** of ammoniacal nitrogen in soils
INVENTOR(S): Goya, Hirohito; Nakanishi, Michio; Saruwatori, Kenichi; Hirose, Akira; Shinozawa, Tetsuichi
PATENT ASSIGNEE(S): Yoshitomi Pharmaceutical Industries, Ltd.
SOURCE: Jpn. Tokkyo Koho, 6 pp.
CODEN: JAXXAD
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 47004966	B4	19720212	JP 1969-54885	19690711

GI For diagram(s), see printed CA Issue.
AB Sulfanilamide derivs. (I or II), where X is -H, -COCH3, -C(:NH)NH2, 2-pyridinyl, 2-**pyrimidinyl**, and 2-pyrazinyl, R is H or a half amide of a C2-C8 dicarboxylic acid and R1 is 1,2-benzendiyl or (CH2)n (n = 0-6), were active as nitrification **inhibitors**. They can be applied together with the fertilizer or after fertilizer application and remain active up to the next growth period.

L9 ANSWER 40 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1973:83310 CAPLUS
DOCUMENT NUMBER: 78:83310
TITLE: Sulfanilamide **oxidation inhibitors** for ammoniacal nitrogen in soils
INVENTOR(S): Goya, Hirohito; Hidaka, Nobuhiro; Hirose, Akira; Shinozawa, Tetsuichi
PATENT ASSIGNEE(S): Mitsui Toatsu Chemicals Co., Ltd.
SOURCE: Jpn. Tokkyo Koho, 5 pp.
CODEN: JAXXAD
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 47004961	B4	19720212	JP 1968-22045	19680405

GI For diagram(s), see printed CA Issue.

AB New sulfanilamide nitrification **inhibitors** were developed; they have a general structure I, where Y may be H, 2-pyridinyl, 4-methyl-2-thiazolyl, 1,3,4-thiadiazolyl, 2-**pyrimidinyl**, amidino, benzoyl, 4,6-dichloro-s-triazin-2-yl, or 4,6-diamino-s-triazin-2-yl.Z is C2-6 alkyl, and R and R1 are H or Me. Good results were obtained with 1-150 ppm **inhibitor** concns. in soil. The **inhibitors** can be applied before, at, or after org. N fertilizer is introduced to the soil and they remain effective also in the next growing season. The **inhibitors** can be applied to the soil surface and then plowed into soil or by spray or as an emulsion.

L9 ANSWER 41 OF 73 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1973:15263 CAPLUS
DOCUMENT NUMBER: 78:15263
TITLE: **Oxidation** of diarylmethylpyridine and **pyrimidine** carbanions. Steric requirements
AUTHOR(S): Kress, Thomas J.; Moore, Larry L.
CORPORATE SOURCE: Lilly Res. Lab., Eli Lilly and Co., Indianapolis, Indiana, USA
SOURCE: J. Heterocycl. Chem. (1972), 9(5), 1161-4
CODEN: JHTCAD
DOCUMENT TYPE: Journal
LANGUAGE: English

GI For diagram(s), see printed CA Issue.

AB Oxidn. of 2-,3-, and 4-(diphenylmethyl)pyridine, 5-(diphenylmethyl)-**pyrimidine**, and 5-[(2,4-dichlorophenyl)phenylmethyl] **pyrimidine** (I; R = Cl, R1 = R2 = R3 = H) by NaOH and O in Me2SO gave 72-97% triaryl carbinols. The following I did not react: R = R2 = H, R1 = R3 = Cl; R = R1 = Cl, R2 = R3 = H; R = R1 = R3 = Cl, R2 = H; r = R1 = R2 = Cl, R3 = H. Thus, the steric shielding imposed on the central C atom of the carbanion intermediate by 2 axially opposed Cl atoms **inhibited** an effective collision with O.

L9 ANSWER 42 OF 73 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1972:98388 CAPLUS
DOCUMENT NUMBER: 76:98388
TITLE: Oxidations of nucleic acids and their components in soil. I. **Oxidation** in air-dried soil samples
AUTHOR(S): Drobnikova, Vera
CORPORATE SOURCE: Dep. Plant Physiol. Soil Biol., Charles Univ., Prague, Czech.
SOURCE: Zentralbl. Bakteriol., Parasitenk., Infektionskr. Hyg., Abt. 2 (1971), 126(7), 700-12
CODEN: ZBPIA9
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The degree of oxidn. of RNA was lower than that of its individual components. With storage, the respiration of air-dried soil increased, but the content of NO3- changed very little. Ribose oxidn. was lower than that of glucose. N and P, each, increased glucose oxidn. and nitrification in soil. NH4)2CO3 or purine or **pyrimidine** bases depressed the respiration rate during the 1st hr even in the presence of glucose. Greater oxidn. occurred with purines than with glucose. Hypoxanthine was oxidized 1st, followed by adenine and uric acid. The oxidn. of **pyrimidines** began earlier but at a lower rate than that of purines. Addn. of glucose shortened the primary oxidn. of purines and **pyrimidines**, but the rate of oxidn. remained unchanged. In the oxidn. of purine and **pyrimidine** ribosides, the sugar moiety

was probably oxidized 1st, followed by oxidn. of the bases.

L9 ANSWER 43 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1971:135921 CAPLUS
DOCUMENT NUMBER: 74:135921
TITLE: Xanthine oxidase-mediated **oxidation** of epinephrine
AUTHOR(S): Valerino, Donald M.; McCormack, John J.
CORPORATE SOURCE: Coll. Med., Univ. Vermont, Burlington, Vt., USA
SOURCE: Biochem. Pharmacol. (1971), 20(1), 47-55
CODEN: BCPCA6
DOCUMENT TYPE: Journal
LANGUAGE: English
GI For diagram(s), see printed CA Issue.
AB Epinephrine bitartrate (I) stimulation of purines oxidn. by xanthine oxidase (XO) was attributable to concomitant oxidn. of I as well as purine substrate, hypoxanthine (II), by XO prepns. XO did not oxidize I under the conditions employed in the absence of an oxidizable substrate such as II. The oxidn. of I in the presence of XO and II was **inhibited** strongly by allopurinol (4-hydroxypyrazolo-(3,4-d)**pyrimidine**). The product of I oxidn. in the XO system was adrenochrome (2,3-dihydro-3-hydroxy-N-methylindole-5,6-quinone).

L9 ANSWER 44 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1969:446108 CAPLUS
DOCUMENT NUMBER: 71:46108
TITLE: **Oxidation** of some aminopteridines by xanthine oxidase
AUTHOR(S): Valerino, Donald M.; McCormack, John J.
CORPORATE SOURCE: Coll. of Med., Univ. of Vermont, Burlington, Vt., USA
SOURCE: Biochim. Biophys. Acta (1969), 184(1), 154-63
CODEN: BBACAQ
DOCUMENT TYPE: Journal
LANGUAGE: English
AB 2-Aminopteridine and its 4- and 7-monohydroxylated derivs. were efficiently oxidized at pH 7.4 and 37.degree. by milk xanthine oxidase to a product identified as 2-amino-4,7-dihydroxypteridine (isoxanthopterin) on the basis of spectroscopic and chromatographic data. The 7-methyl and the 6,7-dimethyl derivs. of 2-aminopteridine were rapidly oxidized by xanthine oxidase, but 4-methyl-2-aminopteridine did not appear to be an effective substrate for the enzyme. 4-Aminopteridine and its 2- and 7-monohydroxy derivs. were converted into 4-amino-2,7-dihydroxypteridine by xanthine oxidase. 4-Aminopteridine and its 6,7-dimethyl deriv. were oxidized considerably less rapidly than the corresponding 2-aminopteridines. The order of susceptibility of the aminopteridines (2- > 4-) to oxidn. by xanthine oxidase was different from that previously reported by Bergmann and Kwietny for the analogous hydroxypteridines (4- > 2-). 4-Hydroxypyrazolo-(3,4-d)-**pyrimidine** (allopurinol) strongly **inhibited** the oxidn. of the aminopteridines by xanthine oxidase.

L9 ANSWER 45 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1969:413086 CAPLUS
DOCUMENT NUMBER: 71:13086
TITLE: Role of alkyl substitution in 2,3-disubstituted and 3-substituted 4-quinazolones on the **inhibition** of pyruvic acid **oxidation**
AUTHOR(S): Parmar, Surendra S.; Kishor, K.; Seth, P. K.; Arora, R. C.
CORPORATE SOURCE: King George's Med. Coll., Lucknow Univ., Lucknow, India
SOURCE: J. Med. Chem. (1969), 12(1), 138-41
CODEN: JMCMAR

DOCUMENT TYPE: Journal
LANGUAGE: English
GI For diagram(s), see printed CA Issue.
AB Several 2,3-disubstituted and 3-substituted 4-quinazolones I (R = Me and H; R1 = Et and Me2) were synthesized to investigate structure-activity relation of these quinazolones with respect to their ability to **inhibit** pyruvic acid oxidn. by rat brain homogenate. 2-Methyl-3-(o-tolyl)-4-quinazolone was used for comparison. In general, 2,3-disubstituted quinazolones exhibited greater **inhibitory** properties as compared to the corresponding 3-substituted quinazolones. Introduction of the alkyl substituent(s) on the phenyl nucleus, attached to the 3 position of the quinazolone mol., significantly influenced the enzyme **inhibitory** properties of these quinazolones. In both series, max. **inhibition** of the oxidn. of pyruvic acid was observed with quinazolones synthesized from 2,4-dimethylaniline. In-crease in the concn. of the quinazolones simultaneously increased the enzyme **inhibition**. Added NAD, responsible for the increase in the respiratory activity of brain homogenate during oxidn. of pyruvic acid, reduced the **inhibition** produced by 2,3-disubstituted and 3-substituted 4-quinazolones.

L9 ANSWER 46 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1968:424726 CAPLUS
DOCUMENT NUMBER: 69:24726
TITLE: Enzymic decomposition of urocanic acid. VII. Identification of the enzyme catalyzing the **oxidation** of 4(5)-imidazolone-5(4)-propionic acid as an aldehyde oxidase
AUTHOR(S): Payes, Benjamin; Greenberg, David M.
CORPORATE SOURCE: Sch. of Med., Univ. of California, San Francisco, Calif., USA
SOURCE: Arch. Biochem. Biophys. (1968), 125(3), 911-17
CODEN: ABBIA4
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The enzyme from guinea pig liver that catalyzes the oxidn. of 4(5)-imidazolone-5(4)-propionic acid to hydantoin-5-propionic acid has properties similar to those described for aldehyde oxidase (or so-called xanthine dehydrogenase). The guinea pig liver enzyme catalyzes oxidn. of aldehydes, purines, a **pyrimidine**, and a no. of other N-contg. heterocyclic compds. The ratio of enzyme activity on imidazolone propionate and 4-hydroxypyrimidine remained reasonably const. over a 130-fold enrichment of enzyme purity. The guinea pig liver enzyme responded similarly to activators and **inhibitors** of the aldehyde oxidases from other sources. The enzyme resembles the so-called xanthine dehydrogenase in that it catalyzes oxidn. of purine to 8-hydroxypurine, not 6-hydroxypurine. Utilization of O for the oxidn. of hypoxanthine and xanthine is very sluggish, but their oxidn. is markedly accelerated by other electron acceptors, e.g., 2,6-dichlorophenolindophenol, as is the case with xanthine dehydrogenase.

L9 ANSWER 47 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1967:450375 CAPLUS
DOCUMENT NUMBER: 67:50375
TITLE: **Oxidation** of **pyrimidine** nucleosides and nucleotides by osmium tetroxide
AUTHOR(S): Burton, Kenneth
CORPORATE SOURCE: Massachusetts Gen. Hosp., Boston, Mass., USA
SOURCE: Biochem. J. (1967), 104, 686-94
CODEN: BIJOAK
DOCUMENT TYPE: Journal
LANGUAGE: English
AB **Pyrimidine** nucleosides such as thymidine, uridine, or cytidine

are oxidized readily at 0.degree. by osmium tetroxide in NH4Cl buffer. There is virtually no oxidn. in bicarbonate buffer of similar pH. Oxidn. of 1-methyluracil yields 5,6-dihydro-4,5,6-trihydroxy-1-methyl-2-pyrimidone. Osmium tetroxide and ammonia react reversibly in aq. soln. to form a yellow 1:1 complex, probably OsO3NH. A second mol. of ammonia must be involved in the oxidn. of UMP since the rate of this reaction is approx. proportional to the square of the concn. of unprotonated ammonia. 4-Thiouridine reacts with osmium tetroxide much more rapidly than does uridine. The changes of absorption spectra are different in NaHCO3 buffer and in NH4Cl buffer. They occur faster in the latter buffer and, under suitable conditions, cytidine is a major product. Poly(uridylic acid) is oxidized readily by ammoniacal osmium tetroxide, but its oxidn. is **inhibited** by poly(adenylic acid). **Pyrimidines** of yeast amino acid-transfer RNA are oxidized more slowly than the corresponding mononucleosides, esp. the thymine residues. Appreciable oxidn. can occur without change of sedimentation coeff.

L9 ANSWER 48 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1967:9261 CAPLUS
DOCUMENT NUMBER: 66:9261
TITLE: Possible role of xanthine oxidase in the **oxidation** of glyoxylate to oxalate
AUTHOR(S): Gibbs, Dorothy A.; Watts, Richard W. E.
CORPORATE SOURCE: St. Bartholomew's Hosp., London, Engl.
SOURCE: Clin. Sci. (1966), 31(2), 285-97
CODEN: CSCIAE
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Xanthine oxidase catalyzed the oxidn. of glyoxylate to oxalate, but played only a minor role in the overall production of oxalate in the intact human. The Km for milk xanthine oxidase with respect to glyoxalate was 6 .times. 10-4M, and the reaction was **inhibited** by allopurinol (4-hydroxypyrazolo-[3,4-d]**pyrimidine**) and by pteridylaldehyde (2-hydroxy-4-amino-6-formylpteridine), but not by disulfiram (tetraethylthiuram disulfide). The oxidn. of glyoxylate in the supernatant fraction of human liver tissue was less susceptible to **inhibition** by allopurinol and pteridylaldehyde than the corresponding fraction from rat tissue. Studies on persons with xanthinuria showed that oxalate excretion was not abolished when xanthine oxidase was congenitally absent. 37 references.

L9 ANSWER 49 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1966:431470 CAPLUS
DOCUMENT NUMBER: 65:31470
ORIGINAL REFERENCE NO.: 65:5867g-h
TITLE: New aspects of the **oxidation** of glycolate in etiolated plants
AUTHOR(S): Scoppa, P.; Tafuri, F.
CORPORATE SOURCE: Univ. Perugia, Italy
SOURCE: Ann. Fac. Agrar. Univ. Studi, Perugia (1964), 19, 99-121
DOCUMENT TYPE: Journal
LANGUAGE: Italian
AB Green wheat (Thatcher variety) plants contain an active glycolic acid oxidase (I), while in etiolated plants I activity is practically negligible. Adding FMN to slurries of green or etiolated plants produces an increase of I activity, a final concn. of 10-4M being necessary for the max. effect. The addn. of FMN produces an increase of enzyme activity equal to .apprx.10-fold the initial value in etiolated plants and .apprx.2.5-fold in green plants. .alpha.-Hydroxy-2-**pyrimidinemethanesulfonate** and the hydrazide of isonicotinic acid, **inhibitors** of I, do not affect activation due to the addn. of FMN to etiolated plants. If glycolate is added to slurries of etiolated

plants, or the slurries are maintained at ambient temps., notable changes in absorbance are observed; this does not happen with slurries of green plants.

L9 ANSWER 50 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1966:430660 CAPLUS
DOCUMENT NUMBER: 65:30660
ORIGINAL REFERENCE NO.: 65:5714e-f
TITLE: Antioxidative effects of purine bases on hydrogen peroxide **oxidation** of **pyrimidine** bases
AUTHOR(S): Melzer, M. S.; Tomlinson, R. V.
CORPORATE SOURCE: State Univ. of New York, Buffalo
SOURCE: Arch. Biochem. Biophys. (1966), 115(1), 226-9
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Guanylic, polyadenylic, and adenylic acids were tested for their abilities to **inhibit** H2O2 oxidn. of the base moieties of polycytidylic and polyuridylic acids. Guanylic acid protected both of these substrates, but adenylic acid protected neither. Polyadenylic acid showed antioxidative capacity only when complex formation could occur, i.e., in the presence of polyuridylic acid. The chelating agent, EDTA, was as effective as guanylic acid in protecting both **pyrimidine** substrates.

L9 ANSWER 51 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1961:54900 CAPLUS
DOCUMENT NUMBER: 55:54900
ORIGINAL REFERENCE NO.: 55:10581g-i,10582a-c
TITLE: Deoxyribonucleic acid (DNA) synthesis, respiration, and virulence in pneumococci
AUTHOR(S): Firshein, W.
CORPORATE SOURCE: Wesleyan Univ., Middletown, CT
SOURCE: Ann. N.Y. Acad. Sci. (1960), 88, 1054-74
DOCUMENT TYPE: Journal
LANGUAGE: Unavailable
AB Selective stimulation of DNA synthesis occurred in resting cells of type I and type II virulent pneumococci (I) supplemented with enzymic DNA digests, all of the naturally occurring deoxynucleosides and deoxynucleotides, and the 4 nucleoside diphosphates derived from ribonucleic acid (RNA). Cells of the avirulent forms (II) did not respond appreciably to these supplements. Characteristic of the enhanced DNA synthesis with I was a decrease in synthetic activity from the max. levels after 30 or 50 min. A supplement contg. the enzymic DNA digest, Mn++, and purines and **pyrimidines** also stimulated DNA synthesis by virulent pneumococci, but the magnitude was not as great as with the nucleoside deriv. supplement; the mechanism of action appeared to be different. The Mn++-contg. supplement enhanced DNA, RNA, and protein syntheses; the nucleoside deriv. affected DNA synthesis primarily. The active material from the enzymic DNA digest was not identical with known breakdown products and apparently acted other than as a mere contributor of DNA precursors. The optimum supplementation system also elicited a striking selective stimulation of O uptake in I contg. glucose and **inhibited** O uptake when II was present. In the absence of glucose I was incapable of oxidizing the nucleic acid breakdown products, whereas cells of II exhibited definite activity. A substantial amt. of the sugar was assimilated in both suspensions when the extracellular supplements were absent. Some of this assimilated glucose was oxidized in suspensions of I when the nucleic breakdown products were added. A substance contg. twice as much N as that found in the entire extracellular nucleic acid breakdown supplement did not enhance O uptake in I to any degree. The deoxynucleotide mixt. and the enzymic DNA digest in combination produced the greatest stimulation of O consumption. The deoxynucleoside and the nucleoside diphosphate mixts. exerted a depressing effect on the max.

levels obtained with these other supplements. Any supplement contg. the enzymic DNA digest stimulated O uptake to a greater extent than such supplements lacking the digest. High concns. of single deoxynucleosides, except for deoxycytidine which **inhibits** respiration, enhanced oxidn. of glucose almost as well as the full supplement. Mn++ stimulated glucose oxidn. in I while **inhibiting** such **oxidation** in II.

L9 ANSWER 52 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1959:40050 CAPLUS
DOCUMENT NUMBER: 53:40050
ORIGINAL REFERENCE NO.: 53:7216e-h
TITLE: Derivatives of 6-methyl-1,2,4-triazine
PATENT ASSIGNEE(S): Wellcome Foundation Ltd.
DOCUMENT TYPE: Patent
LANGUAGE: Unavailable
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
GB 802122		19581001	GB	

GI For diagram(s), see printed CA Issue.
AB The triazines N:CY.CMe:N.N:CX (I), where X may be HO, HS, RS, or RNH, and Y may be HS or H2N may be prepd. by reactions with P2S5 or NH3, **oxidation** with KMnO4 or alkylation with Me2SO4. Freshly-ground P2S5 (30 g.) and 100 ml. Tetralin were added to 10 g. I (X = Y = OH), the mixt. was stirred at 180-190.degree. for 3 hrs., cooled, filtered, the ppt. washed with petr. ether and decompd. with 250 ml. H2O, and the aq. soln. worked up with ether to give orange plates of I (X = Y = HS) (II), m. 217-18.degree. (boiling H2O). II treated at 122.degree. in a sealed tube with alc. NH3 (satd. at 0.degree.) gave quant. yields of I (X = HS, Y = H2N) (III), beige needles repptd. from cold 0.1N HCl with cold 0.1N NaOH, darkened at 270.degree., did not m. at 310.degree.. III (10 g.) was oxidized with 25 g. KMnO4 in 500 ml. H2O, filtered and the filtrate concd. to dryness. The K sulfonate was dissolved in 20 ml. H2O, brought to pH 1 with 0.1N HCl with cooling, and the mixt. kept at room temp. 2 days, and neutralized to pH 7 with dil. NH4OH to give I (X = OH, Y = H2N), decomp. 327.degree.. Me2SO4 (3.4 ml.) added with shaking over 30 min. to 5 g. III in 100 ml. 0.35N NaOH gave I (X = MeS, Y = H2N) (IV), plates, m. 164-5.degree. (MeOH-ether-petr. ether). IV (5 g.) was heated in a bomb 72 hrs. at 155.degree. with 250 ml. EtOH satd. at 0.degree. with NH3 to give beige crystals of I (X = Y = H2N), m. 234-8.degree. (EtOH) (decompn.). These compds. are useful as antimetabolites of the isosteric **pyrimidine** derivs., microorganism **inhibitors**, and pharmaceutical intermediates.

L9 ANSWER 53 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1958:116355 CAPLUS
DOCUMENT NUMBER: 52:116355
ORIGINAL REFERENCE NO.: 52:20662c-d
TITLE: The metabolism of pyrazolo[3,4-d]**pyrimidines** by the rat
AUTHOR(S): Feigelson, Philip; Davidson, Jack D.
CORPORATE SOURCE: Columbia Univ.
SOURCE: Cancer Research (1958), 18, 226-8
DOCUMENT TYPE: Journal
LANGUAGE: Unavailable
GI For diagram(s), see printed CA Issue.
AB The recent development of a new series of carcinostatic purine analogs, the pyrazolo[3,4-d]**pyrimidines**, led to their study as substrates and **inhibitors** of certain purine **oxidation** systems in vitro. In the present study, pyrazoloadenine was found to be oxidized in

the rat to pyrazoloisoguanine. The rat metabolizes pyrazolo[3,4-d] **pyrimidines** as follows:

L9 ANSWER 54 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1958:61603 CAPLUS
DOCUMENT NUMBER: 52:61603
ORIGINAL REFERENCE NO.: 52:11153i,11154a
TITLE: Subtle interactions of cupric ion with nucleic acid and components
AUTHOR(S): Frieden, Earl; Alles, Jeanne
CORPORATE SOURCE: Florida State Univ., Tallahassee
SOURCE: J. Biol. Chem. (1958), 230, 797-804
DOCUMENT TYPE: Journal
LANGUAGE: Unavailable
AB cf. C.A. 51, 11424a. Subtle interactions between Cu(II) ions and nucleic acids and their components were studied by their **inhibition** of the Cu(II)-catalyzed **oxidation** of ascorbate. The effectiveness of Cu(II) chelation was purine > purine nucleotide = ribonucleic acid-deoxyribonucleic acid > purine nucleoside > **pyrimidine** nucleotide. The deoxyribose derivs. were uniformly stronger Cu(II) chelators. The structure of these chelates and their possible biol. significance are discussed.

L9 ANSWER 55 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1957:86264 CAPLUS
DOCUMENT NUMBER: 51:86264
ORIGINAL REFERENCE NO.: 51:15661g-i
TITLE: Pyrazolopyrimidines as **inhibitors** and substrates of xanthine oxidase
AUTHOR(S): Feigelson, Philip; Davidson, J. D.; Robins, Roland K.
CORPORATE SOURCE: Columbia Univ.
SOURCE: J. Biol. Chem. (1957), 226, 993-1000
DOCUMENT TYPE: Journal
LANGUAGE: Unavailable
AB cf. C.A. 50, 16911i, 13036c. The **inhibitor** and substrate specificities of xanthine oxidase were studied by use of purine analogs, pyrazolo(3,4-d)**pyrimidines** (loc. cit.). Pyrazoloisoguanine competitively **inhibits** the enzyme; 50% **inhibition** occurs at 10-6M. Pyrazoloadenine is less **inhibitory**; 50% **inhibition** is seen at 10-4M. Methylation of the amino or ring N atoms still further decreases **inhibition**. Xanthine oxidase catalyzed the **oxidation** of pyrazoloadenine to pyrazoloisoguanine, which was characterized by paper chromatography, ion exchange, and spectrophotometry. The possible relation between the **inhibition** in vitro of xanthine oxidase and the carcinostatic activities of these compds. is discussed (cf. C.A. 50, 483b).

L9 ANSWER 56 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1957:57304 CAPLUS
DOCUMENT NUMBER: 51:57304
ORIGINAL REFERENCE NO.: 51:10642g-i,10643a
TITLE: Studies on the influence of cobalt chloride on the growth of actinomycetes. I
AUTHOR(S): Kojima, Hisao; Matsuki, Midori
CORPORATE SOURCE: Tohoku Univ., Sendai
SOURCE: Tohoku J. Agr. Research (1956), 7, 175-87
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Twelve strains of actinomycetes were cultured in Czapek's medium contg. 0, 2, or 8 .gamma. of CoCl2.6H2O (I)/ml. The presence of I in the media, at a concn. of 2 .gamma./ml. resulted in complete growth **inhibition** of 8 of these strains. When one of these strains (K300) was cultured in a glucose-bouillon medium it was found that concns. of I as high as

100.gamma./ml. were tolerated. The rate of glucose **oxidation** of this strain when cultured on Czapek's medium was **inhibited** by I. The **inhibitory** effect of Co on the glucose **oxidation** rate of the K300 strain cultured in the glucose-bouillon medium was greater than that observed in the organisms grown in the Czapek medium. A strain (346) which showed no growth **inhibition** when I was added to the media also showed less **inhibition** of the glucose **oxidation** rate in the presence of I. Casein hydrolyzate added to media which contained concns. of I as high as 12 .gamma./ml. resulted in a reversal of the growth **inhibition**. **Pyrimidine** and purine bases were only slightly effective in reversing growth **inhibition**. When L-histidine or L-cysteine was added to the media contg. Co (80 .gamma./ml.) the growth **inhibition** due to Co was reversed. **Inhibition** of the glucose **oxidation** rate by Co did not occur when these compds. were present initially. Preincubation of the organisms in media contg. L-cysteine resulted in nearly complete reduction of the Co **inhibition** of the glucose **oxidation** rate.

L9 ANSWER 57 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1956:16740 CAPLUS
DOCUMENT NUMBER: 50:16740
ORIGINAL REFERENCE NO.: 50:3540a-c
TITLE: Effect of purine and **pyrimidine** analogs on enzyme induction in Mycobacterium tuberculosis
AUTHOR(S): Ottey, Leo
CORPORATE SOURCE: Duke Univ., Durham, NC
SOURCE: J. Pharmacol. Exptl. Therap. (1955), 115, 339-42
DOCUMENT TYPE: Journal
LANGUAGE: Unavailable
AB Addn. to the medium of 6-mercaptopurine, 2,6-diaminopurine, 5-aminouracil, 5-methyl-2-thiouracil, 6-methyl-2-thiouracil, 2-thiocytosine, or 2-thiouracil **inhibited** the formation of adaptive enzymes for the **oxidation** of benzoic acid by M. tuberculosis; 2-thioorotic acid had no effect. The first 5 compds. and also 2-thioorotic acid **inhibited** formation of the adaptive enzymes for the **oxidation** of myo-inositol; 2-thiouracil had no effect here.

L9 ANSWER 58 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1955:73412 CAPLUS
DOCUMENT NUMBER: 49:73412
ORIGINAL REFERENCE NO.: 49:13900h-i,13901a-b
TITLE: Action of some derivatives of **pyrimidine** in the **oxidation** of pyrocatechol
AUTHOR(S): Mkhitaryan, V. G.; Shukuryan, S. G.; Avakimova, E. A.
SOURCE: Doklady Akad. Nauk Armyan. S.S.R. (1953), 17(No. 3), 81-5
DOCUMENT TYPE: Journal
LANGUAGE: Russian
AB 4-Methyl-2-thiouracil (I) in phosphate buffer at pH 7.26 strongly **inhibits** the **oxidation** of pyrocatechol (II) either in the presence or the absence of Cu++ or Fe+++ ions. In phosphate buffer at pH 6.4 I alone or in the presence of Cu++ ion strongly **inhibits** the **oxidation** of II, but it does not **inhibit** the **oxidation** in presence of Fe+++ ion. 4-Methyl-uracil (III) in phosphate buffer at pH 7.23 weakly **inhibits** the **oxidation** of II, but it shows little influence on the **oxidation** in the presence of Fe+++ and Cu++ ions. In a phosphate buffer at pH 6.37 III does not **inhibit** the **oxidation** of II alone or in the presence of Fe+++ ion, but it does **inhibit** in the presence of Cu++ ion. The tests were carried out with II which had been twice-recrystd. from phosphate buffer at pH 7.2 and pH 6.4. The **oxidation** of II is measured manometrically by the quantity of O

consumed in a Warburg app. at 37.degree.. In the Warburg respirometer were used 3.3-ml. samples (7 mg. II in 1 ml. soln., 5 mg. I in 2.1 ml. of soln., and 0.2 ml. 30% KOH). In the tests with Cu and Fe ions (used as their sulfates), 3.1-ml. samples contg. 0.3 mg. Fe (0.06 ml.) or 0.003 mg. Cu (0.06 ml.) were employed.

L9 ANSWER 59 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1955:43135 CAPLUS
DOCUMENT NUMBER: 49:43135
ORIGINAL REFERENCE NO.: 49:8339a-e
TITLE: Second type of bacterial thiaminase
AUTHOR(S): Fujita, Akiji; Nose, Yoshitsugu; Kuratani, Kazuo
CORPORATE SOURCE: Kyoto Prefectural Univ.
SOURCE: J. Vitaminol. (Japan) (1954), 1, 1-7
DOCUMENT TYPE: Journal
LANGUAGE: Unavailable

AB The properties of thiaminase II (I) of Bacillus aneurinolyticus were different from thiaminase from Bacillus thiaminolyticus, shellfish, fish, and plant sources, called thiaminase I (II). The effects of various amines (III) on I (pH 7, 60.degree., with a final concn. of III of 10-3M) by use of the same method as was used on II by F., et al. (C.A. 46, 12281h), showed I not to be activated and in many cases **inhibited** by III, contrary to the findings on II. With pyridine and quinoline the fluorescent substance (pyrichrome and quinochrome) did not appear after ferricyanide **oxidation**, showing the lack of the base-exchange reaction. Enzymic action of I on thiamine produced **pyrimidinemethanol** (IV) and the thiazole moiety (V), whereas II never gave IV and the fate of the **pyrimidine** moiety could not be traced in this case by the Dragendorff reagent. Since I was not activated by various III, the base-exchange reaction was not expected. However, the formation of **pyrimidinemethylaniline** (VI) by I in the presence of thiamine and aniline was shown as in the case of II. Similar base-exchange reactions with other III did not take place and only the formation of IV was demonstrated. A study of various molar ratios of aniline to thiamine in the formation of VI showed that IV formed in low aniline concn. while VI formed in aniline concn. higher than twice that of thiamine. It was observed that thiamine was formed as the reverse reaction of thiaminase by incubating II with the base-exchanged **pyrimidine** deriv. and the thiazole moiety of thiamine (loc. cit.). I produced a reverse reaction when incubated with VI, contrary to the case of II. It seems probable from the above findings to assume that I itself does not catalyze the base-exchange reaction, but that a second enzyme accompanying it catalyzes the reaction which makes the reaction appear to be a base exchange. Furthermore, since the base-exchange reaction does not take place with other III, but aniline, makes the assumption of a special enzyme probable.

L9 ANSWER 60 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1954:43494 CAPLUS
DOCUMENT NUMBER: 48:43494
ORIGINAL REFERENCE NO.: 48:7790f-h
TITLE: **Oxidation** of ascorbic acid by Terramycin
AUTHOR(S): Dudani, A. T.; Krishnamurti, C. R.
CORPORATE SOURCE: Central Drug Research Inst., Lucknow, India
SOURCE: Biochim. et Biophys. Acta (1954), 13, 505-9
DOCUMENT TYPE: Journal
LANGUAGE: Unavailable

AB Terramycin (I) (and dihydrostreptomycin to a very much lesser degree) accelerated the rate of **oxidation** of ascorbic acid (II). Aureomycin, neomycin, and penicillin, on the other hand, **inhibited oxidation**, penicillin G exerting the strongest effect. I did not cause **oxidation** of any of a large no. of reducing agents, suggesting that the action of I is specific for II. The effect of I was

proven not to be caused by Cu impurity. The catalytic activity of I was found to be thermostable, to not affect either antibacterial activity or fluorescence of Iin the ultraviolet, to have a pH optimum of 8, and to be present in amts. as low as 1 .gamma. of I. Penicillin G and 8-hydroxyquinoline **inhibited** the activity of I, but a variety of purines, **pyrimidines**, amino acids, vitamins, HCN, etc. had no effect. Many of the substances, however, **inhibited** Cu catalysis.

L9 ANSWER 61 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1954:42669 CAPLUS
DOCUMENT NUMBER: 48:42669
ORIGINAL REFERENCE NO.: 48:7670a-b
TITLE: The **inhibitors** of the **oxidation** of L-ascorbic acid. The mechanism of action of thiamine
AUTHOR(S): Gero, Etienne
SOURCE: Compt. rend. (1954), 238, 959-61
DOCUMENT TYPE: Journal
LANGUAGE: Unavailable
AB cf. C.A. 47, 2781c. Cuprothiamine contains 2 atoms of Cu per mol. of thiamine. The thiazole nucleus (I) strongly **inhibits** the **oxidation** of L-ascorbic acid by Cu++ ions. The **pyrimidine** nucleus (II), an **inhibitor** by itself, diminishes the action of I when I is assocd. with II in the same mol.

L9 ANSWER 62 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1954:32707 CAPLUS
DOCUMENT NUMBER: 48:32707
ORIGINAL REFERENCE NO.: 48:5890h-i,5891a
TITLE: The action of 4-methyluracil and 4-methyl-2-thiouracil on the **oxidation** process of ascorbic acid
AUTHOR(S): Mkhitaryan, V. G.; Avakimova, E. A.; Shchukuryan, S. G.
CORPORATE SOURCE: Sci. Research Inst. Roentgenol. and Oncol., Ministry Health Armenian S.S.R., Erevan
SOURCE: Voprosy Pitaniya (1953), 12(No. 4), 23-8
DOCUMENT TYPE: Journal
LANGUAGE: Unavailable
AB The **oxidation** of ascorbic acid (I) solns. was detd. manometrically in a Warburg respirometer at 37.degree. and by indophenol titration. 4-Methyl-2-thiouracil (II) **inhibits** the **oxidation** of I more effectively in phosphate buffer at 6.24 than at pH 7.23. This compd. **inhibits** the **oxidation** of I in the presence of Cu ions at pH 6.24 and is as strong an antioxidant as uric acid. The antioxidative action of II in the presence of Fe ions is much weaker. 4-Methyluracil alone shows some **inhibitory** action on the **oxidation** process of I at pH 6.24, but does not depress the **oxidation** in the presence of Fe and Cu ions at this pH. II shows stronger **inhibitory** action on the **oxidation** process of I than 4-methyluracil. The mechanism of II action on the **oxidation** of I is believed to be due to the action of the **pyrimidine** ring and to the bivalent S.

L9 ANSWER 63 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1952:36146 CAPLUS
DOCUMENT NUMBER: 46:36146
ORIGINAL REFERENCE NO.: 46:6172c-g
TITLE: The structure of nucleic acids. II. Investigation of pentose nucleic acid and enzyme-resistant residues
AUTHOR(S): Cavalieri, Liebe F.; Kerr, Stanley E.; Angelos, Alice
CORPORATE SOURCE: Sloan-Kettering Inst., New York, NY
SOURCE: J. Am. Chem. Soc. (1951), 73, 2567-78
DOCUMENT TYPE: Journal

LANGUAGE: Unavailable

AB cf. C.A. 46, 6089b. The interaction of rosaniline with various fractions of yeast nucleic acid and pentose nucleic acid (PNA) from beef pancreas was studied. The binding sites involve the phosphoric acid groups, about 13% of which are available for binding. On the basis of the similar intrinsic binding consts. and n values (no. of available sites), it is suggested that a similar backbone structure exists among the nucleic acid samples studied. The interaction of rosaniline with the ribonuclease-resistant fractions of yeast PNA was also studied. The binding capacity of the n1-type site (bivalent ion) of the resistant fraction is slightly greater than that of the parent PNA, owing to a decrease in steric **inhibition**. In the case of the ribonuclease-phosphatase-treated nucleic acid, the results of the binding process suggest that only one type of site is involved, which corresponds to the univalent anion type. Correlation of the exptl. pH titration curves with theoretical curves constructed from the known compn. of various samples of nucleic acid, a ribonuclease-resistant fraction and a ribonuclease-phosphatase-resistant fraction indicates that some of the OH groups of guanine and/or uracil are unavailable for titration and may be covalently bound in a phosphate-type bond. In the case of the ribonuclease-phosphatase-treated nucleic acid, purine and **pyrimidine** analyses, periodate **oxidation** titers and ion-exchange analysis provide addnl. evidence for such a covalent linkage. Both PNA and the ribonuclease-resistant fraction were subjected to periodate **oxidation**, and it appears that a D-riboside phosphate other than the 2' or 3' exists in the resistant fraction. Ultraviolet and infrared absorption spectra of both treated and untreated nucleic acids are similar and cannot be used effectively as a means of identification. X-ray powder patterns suggest that some of the residues remaining after the action of ribonuclease and acid-phosphatase are partially cryst.

L9 ANSWER 64 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1952:738 CAPLUS
DOCUMENT NUMBER: 46:738
ORIGINAL REFERENCE NO.: 46:151g-h
TITLE: **Inhibition** of the oxidases of Agaricus campestris
AUTHOR(S): Voinovitch, Igor
CORPORATE SOURCE: Inst. natl. conserve, Paris
SOURCE: Bull. soc. chim. biol. (1951), 33, 337-46
DOCUMENT TYPE: Journal
LANGUAGE: Unavailable

AB cf. C.A. 44, 3540e, 8980b. The **oxidation** in presence of air of pyrogallol and tyrosine by a partially purified soln. of the polyphenol oxidases of A. campestris is **inhibited** more strongly by thiamine than by ascorbic acid. The thiazole moiety of thiamine **inhibits** the **oxidation** of tyrosine and the **pyrimidine** moiety **inhibits** the **oxidation** of pyrogallol. Nicotinic acid **inhibits** the **oxidation** of pyrogallol by the enzyme prepn.; nicotinamide does not. In all the above cases SO2 augments the **inhibiting** action. Cysteine, and glutathione plus SO2, weakly **inhibit** the **oxidation** of pyrogallol. Cystine is inert. Cysteine increases the activity of the succinic dehydrogenase of the mushroom; glutathione is much less active.

L9 ANSWER 65 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1951:19279 CAPLUS
DOCUMENT NUMBER: 45:19279
ORIGINAL REFERENCE NO.: 45:3431b-c
TITLE: **Inhibitory** effect of caffeine and methylene blue on amine oxidase
AUTHOR(S): Ota, Yukito
SOURCE: J. Biochem. (Japan) (1950), 37, 289-99

DOCUMENT TYPE: Journal
LANGUAGE: Unavailable
AB The endogenous respiration of guinea-pig brain pulp at pH 7.6 was **inhibited** 50% by M/500 tyramine (I), and 20% by M/50 caffeine (II); the **inhibition** by I was halved by the simultaneous addn. of M/50 II. Amine oxidase (III) was prepd. by extg. guinea-pig liver with buffer at pH 7.0, followed by dialysis for 5 hrs. The **oxidation** of I by III was **inhibited** by II and by methylene blue. Among the derivs. of purine, **pyrimidine**, and some dyes, the mols. having N-methyl or amino group showed **inhibition** of III.

L9 ANSWER 66 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1950:33621 CAPLUS
DOCUMENT NUMBER: 44:33621
ORIGINAL REFERENCE NO.: 44:6461i,6462a-h
TITLE: Role of copper in the Nadi reaction, and nonprotein models of oxidases and catalase
AUTHOR(S): Pongratz, Edmond
CORPORATE SOURCE: Univ., Geneve, Switz.
SOURCE: Helv. Chim. Acta (1950), 33, 410-17
DOCUMENT TYPE: Journal
LANGUAGE: French
AB To 10 cc. of pH 7.3 phosphate buffer were added 50 .gamma. Cu++ and approx. 0.2 cc. of Nadi mixt. (equal amts. of 0.05 M 1-naphthol in EtOH and either 0.05 M p-C6H4(NH2)2 (I) or the N, N-di-Me deriv. of I in EtOH). The color reaction characteristic of cytochrome oxidase (red or blue, resp.) was given under these conditions, but not when, instead of Cu++, any of 33 other common cations or anions was used. AgNO3 gave an instantaneous gray violet color with the Nadi reagent, and NH4 vanadate weakly catalyzed the **oxidation** of Nadi. The Cu-catalyzed reaction was **inhibited** by Cu-pptg. reagents such as H2S, sol. sulfides, KSCN, and (COOH)2; substances forming complexes with Cu such as dithiooxamide, salicylaldoxime, 8-hydroxyquinoline, cupferron, benzoin oxime, Na diethyldithiocarbamate, dithizone, thiourea, thiouracil, glycine, alanine, D-serine, aspartic acid, HCN, and CO; and oxidizable substances such as ascorbic acid, cysteine, glutathione, and Fe++, which compete with the Nadi reagent and thus forestall its **oxidation**. NH2OH did not fall into this last group, and typical **inhibitors** of dehydrogenases (ICH2COOH, urethan, narcotics, and NaF) did not affect the Cu-catalyzed Nadi reaction. The **inhibitors** cited are, in general, those of true oxidases. The catalytic effect of Cu was increased by many org. compds. contg. tertiary or quaternary N. The most active found were NH3, pyridine, iminazole, **pyrimidine**, and many of their derivs., such as Me3N, chloramine, choline, nicotinic acid, nicotinamide, coramine, aneurine, biuret, uracil, barbiturates, and guanine. CN- acted as an activator when its concn. was less than 1 mole per mole Cu. Its effect was greater when it was added to the Cu after, rather than before, the substrate. The effect of the activators is due to augmentation of the **oxidation** potential of the Cu. As the pH of reaction mixts. contg. Nadi was raised from pH 5, the rate of the Cu-catalyzed **oxidation** of the dimethyl-I at 18.degree. increased until pH 7.2, when it leveled off. The rate of **oxidation** catalyzed by Cu-pyridine kept increasing up to a pH of more than 8. Raising the temp. increased the catalytic effect of Cu complexes, until at a temp. of 80-90.degree. a max. was reached. The soly. of O then became a limiting factor. Cu++, especially in the form of org. complexes, exhibited an oxidase-like action. Cu-iminazole was particularly effective, and oxidized hydroquinone (II), pyrogallol (III), and tincture of guaiac rapidly; and guaicol (IV), catechol (V), resorcinol, orcinol, protocatechuic acid, adrenaline, 1-naphthol, and similar compds. more slowly. Tyrosine was not attacked, but dihydroxyphenylalanine was oxidized to melanin. Monophenols, p-cresol (VI) and a mixt. of VI with glycine were not attacked. In the presence of H2O2, Cu-iminazole

catalyzed the rapid **oxidation** of II, III, IV, V, phloroglucinol, o-, m-, and p-VI, m- and p-C6H4(NH2)2, benzidine, and related compds. The decompn. of H2O2 by Cu(NH3)4++ was much greater than that by Cu++, and was enhanced by adsorption of the catalyst on chalk. Mn++-nicotine complex adsorbed on chalk was also effective. In addn. to the value of these complexes as models of enzymes, they may prove useful for Cu analysis, because of the specificity of the reaction of Cu with the Nadi reagent.

L9 ANSWER 67 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1949:17545 CAPLUS
DOCUMENT NUMBER: 43:17545
ORIGINAL REFERENCE NO.: 43:3426g-i,3427a-i,3428a-c
TITLE: Some heterocyclic analogs of stilbenes
AUTHOR(S): Brown, Daniel M.; Kon, George A. R.
SOURCE: J. Chem. Soc. (1948) 2147-54
DOCUMENT TYPE: Journal
LANGUAGE: Unavailable

AB Derivs. of 4-aminostilbene are known to be carcinogenic and also to exercise an **inhibitory** effect on the development of transplanted tumors in the rat. 4-Methylpyrimidine (1 g.), 1.5 g. 4-Me2NC6H4CHO (I), and 0.5 g. ZnCl2, heated 1.5 hrs. at 165.degree., give 1.2 g. 4-(4-dimethylaminostyryl)**pyrimidine**, yellow, m. 179.degree. (picrate, purple, m. 195-6.degree., forms black needles at 175.degree.). 2-Hydroxy-4,6-dimethylpyrimidine (II) and 1 mol. I give 2-hydroxy-4,6-bis(4-dimethylaminostyryl)**pyrimidine** (III), purple, with 0.5 mol. H2O, decomp. 316-18.degree. [from (HOC2H4)2O]; the residue from MeOCH2CH2OH gives the monostyryl deriv. (IV), scarlet, m. 253.degree. (decompn.) (Stark and Bogemann, C.A. 4, 2466). I (1.5 g.) and 1.5 g. II.HCl in 20 cc. EtOH and 10 cc. H2O, boiled 4 hrs., give a mixt. of III and IV; 1 drop concd. HCl gives the same mixt. II (1.1 g.), 3 g. I, and 10 drops piperidine in 100 cc. EtOH, refluxed 48 hrs., give III. The crude mixt. of III and IV (2 g.), refluxed 2 hrs. with 10 cc. POCl3, gives 1.2 g. 2-chloro-4-(4-dimethylaminostyryl)-6-methylpyrimidine (V), yellow, m. 176-7.degree., and 0.1 g. 2-chloro-4,6-bis(4-dimethylaminostyryl)**pyrimidine** (VI), red, m. 223-4.degree.; these were sepd. by chromatography on Al2O3. VI (0.1 g.) and 1 cc. piperidine refluxed 3 min. give 0.025 g. of the 2-(1-piperidyl) compd., yellow, m. 223-4.degree.. V (0.52 g.) gives 0.6 g. of the 2-(1-piperidyl) compd. (VII), light yellow, m. 168-9.degree.; 2-(4-morpholinyl) analog (VIIA), pale yellow, m. 155.degree., 100%; 2-cyclohexylamino analog, bright yellow, m. 142-3.degree., 82%; 2-(2-diethylaminoethylamino) analog, yellow, m. 80-1.degree., 65%; 2-diethylamino analog, yellow (from petr. ether) or greenish yellow (from aq. alc.), m. 121-2.degree., 74%; 2-[bis(2-hydroxyethyl)amino] analog, bright yellow, m. 116.degree.. VII (1 g.) in 50 cc. EtOH, hydrogenated over 2% Pd-SrCO3 at room temp./atm. pressure, gives 0.8 g. 2-(1-piperidyl)-4-[2-(4-dimethylaminophenyl)ethyl]-6-methylpyrimidine (VIII), m. 70.degree.. V (1 g.) and 0.085 g. Na in 25 cc. EtOH and 10 cc. C6H6, refluxed 1.5 hrs. on a steam bath, give 0.8 g. 2-ethoxy-4-(4-dimethylaminostyryl)-6-methylpyrimidine, orange, m. 120.degree.. The Cl in V could not be removed by refluxing with Zn in aq. dioxane; catalytic reduction gives the 4-[2-(4-dimethylaminophenyl)ethyl] deriv., m. 59-60.degree.; with piperidine it yields VIII. 2-Hydroxy-4-styryl-6-methylpyrimidine. (1.1 g.) and 6 g. POCl3, refluxed 1 hr., give 0.35 g. of the 2-Cl compd., m. 95.degree.; 2-(1-piperidyl) compd., light yellow, m. 94.degree.. 2-Hydroxy-4,6-distyrylpyrimidine yields the 2-Cl compd., m. 177-8.degree.; 2-(1-piperidyl) compd., light yellow, m. 133.degree.. 2-(1-Piperidyl)-4,6-dimethylpyrimidine (IX) m. 60-1.degree.. 2,6-Di-1-piperidyl-4-methylpyrimidine (X) m. 118.degree.. IX and X failed to react with I under a variety of conditions. 2-Amino-4,6-dimethylpyrimidine (5 g.), refluxed 5 min. with 15 cc. Ac2O, treated with 6 g. I in 5 cc. Ac2O, and refluxed 1 hr., gives 0.2 g. 2-acetamido-4-(4-dimethylaminostyryl)-6-methylpyrimidine, yellow, m. 218-19.degree.. VII and VIIA possess considerable growth-

inhibiting action. 6-Quinolinecarboxaldehyde (XI) (5 g.), 5.8 g. 4-O2NC6H4CH2CO2H, and 2 cc. piperidine, heated 1.5 hrs. at 130-40.degree., give 3 g. 6-(4-nitrostyryl)quinoline (XII), yellow, m. 199-200.degree.; reduction of 2 g. XII with 16 g. SnCl2 in 40 cc. AcOH satd. with HCl (stirred several hrs. at room temp. and heated 4 hrs. on the steam bath) gives 0.87 g. 6-(4-aminostyryl)quinoline (XIII), yellow, m. 214-15.degree.. The 8-isomer of XI (6.75 g.) yields 2.3 g. of the 8-isomer of XII, yellow, m. 171.degree.; reduction of 0.5 g. gives 0.38 g. of the 8-isomer of XIII, yellow, m. 156.degree.. XI (3.64 g.) in 25 cc. C6H6, added to PhCH2MgCl (3.3 g. PhCH2Cl) in 50 cc. ether and refluxed 2 hrs., gives only 0.25 g. 2-phenyl-1-(6-quinolyl)ethanol, m. 129.5-30.degree.. PhCH2CO2Na (0.9 g.), 0.9 g. XI, 5 cc. Ac2O, and 0.2 g. ZnCl2, heated 3 hrs. at 160.degree., give 1.4 g. .alpha.-phenyl-6-quinolineacrylic acid, m. 265.degree.; it could not be decarboxylated. A Skraup synthesis with XI and 4-aminostilbene gave no recognizable product. The diazo compd. from 0.5 g. XIII, decompd. with H3PO2, gives only 10 mg. 6-styrylquinoline, m. 119.degree.. SnCl2 reduction of 6-nitro-6-styrylquinoline yields the 6-NH2 compd., brown, m. 198-9.degree.; Ac deriv., with 1 mol. CHCl3, m. 193.degree.. 6-Nitroquinaldine (XIV) (5 g.), 4 g. I, and 0.2 g. ZnCl2, heated 0.5 hr. at 160.degree., give 7.6 g. 6-nitro-2-(4-dimethylaminostyryl)quinoline, deep purple, m. 248-9.degree., 2 g. of which with 10 g. SnCl2 and 15 cc. fuming HCl, heated 1 hr. on the steam bath, give 1.15 g. of the 6-NH2 compd., brown, m. 251-2.degree.; Ac deriv., orange-yellow, m. 241-2.degree.; Sn and HCl give 2-(2-phenylethyl) quinoline. XIV (2.1 g.), 1.7 g. 4-O2NC6H4CHO, and a little ZnCl2, heated at 170.degree., give 6-nitro-2-(4-nitrostyryl)quinoline, yellow, m. 278.degree.; the 6-NH2 deriv. m. 242-3.degree.; these amines are very sensitive to aerial **oxidation**. 1-Methyltetrahydroquinoline (10 g.), added dropwise to 10.5 g. POCl3 and 9.2 g. PhNMeCHO in 10 cc. C6H6 and kept overnight, gives 5.5 g. 6-formyl-1-methyl-1,2,3,4-tetrahydroquinoline (XV), b15 219-21.degree., m. 28-9.degree.; 1.53 g. XV in 25 cc. C6H6, added to PhCH2MgCl (1.23 g. PhCH2Cl) in 30 cc. ether, refluxed 45 min., and the reaction product in 25 cc. C6H6 refluxed 45 min. with 2 g. P2O5, gives 0.6 g. 6-styryl-1-methyl-1,2,3,4-tetrahydroquinoline, m. 93-4.degree.. 2-Methylthiazole (1 cc.), 1.5 g. I, and 0.5 g. ZnCl2, heated 14 hrs. at 160-70.degree., give 10 mg. 2-(4-dimethylaminostyryl)thiazole, yellow, m. 124.degree.. 2-Methylnaphtho[1,2]thiazole (0.5 g.), 0.4 g. I, and 0.5 g. ZnCl2, heated 1.5 hrs. at 160-80.degree., give 0.46 g. of the 2-(4-dimethylaminostyryl) deriv., yellow, m. 170-1.degree.. 2-Methylbenzothiazole (1 g.), 0.8 cc. I, and 2 drops concd. HCl, heated overnight at 100.degree., give 0.46 g. 2-styrylbenzothiazole, m. 112.degree.. 2-Methylbenzoxazole (XVI) gives an HCl salt, m. 154.degree.. XVI (2 cc.), 2 cc. BzH, and 1 g. anhyd. ZnCl2, heated 6 hrs. at 160.degree., give 1 g. 2-styrylbenzoxazole m. 81-2.degree. (picrate, yellow, m. 163-4.degree.); 2-(4-dimethylaminostyryl) compd., yellow, m. 174-5.degree.. 1-Methylphthalazine (2.5 g.), 2.75 g. I, and 0.75 g. ZnCl2, heated 2 hrs. at 160.degree., give 0.39 g. 1-(4-dimethylaminostyryl)phthalazine, orange, m. 186-7.degree.. Furfuraldehyde (5 g.), 9 g. 4-O2NC6H4CH2CO2H, and 1 cc. piperidine, heated 5 hrs. at 130-40.degree., give 2 g. 2-(2-nitrostyryl)furan (XVII), orange, m. 130-1.degree.; the addn. of p-O2NC6H4N2Cl to 2-furanacrylic acid in Me2CO, followed by AcONa and CuCl2, gives XVII. Catalytic reduction of XVII over Raney Ni gives the azoxy compd., orange, m. 231-2.degree. (decompn.); reduction with Zn and NH4Cl in EtOH gives 2-(4-aminostyryl)furan, m. 104.degree.; Ac deriv. m. 201-2.degree.; reduction under acid conditions gives resinous products.

L9 ANSWER 68 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1946:25719 CAPLUS
DOCUMENT NUMBER: 40:25719
ORIGINAL REFERENCE NO.: 40:5063f-i,5064a-e
TITLE: The synthesis of pterins. II. The physicochemical

properties of thiopterins
AUTHOR(S): Polonovski, Michel; Guinand, Sylvanie; Pesson, Marcel; Vieillefosse, Roger
SOURCE: Bull. soc. chim. (1945), 12, 924-9
DOCUMENT TYPE: Journal
LANGUAGE: Unavailable
GI For diagram(s), see printed CA Issue.
AB cf. C.A. 40, 874.6. Thiopterins of type I do not fluoresce while those of type II show an intense blue or green fluorescence similar to pterin derivs. of types III and IV. When I are oxidized with H_2O_2, the S is replaced by O, and the **oxidation** product exhibits strong fluorescence. In II and in IV, 3 conjugated double bonds are present in the **pyrimidine** ring, and as soon as these double bonds disappear, as in I, the fluorescence also disappears. To study this phenomenon further, the ultraviolet absorption spectra of I (R, R' = H), its **oxidation** product (V), II (R, R' = H), IV (R, R' = H), 2-thio-6-oxo-8,9-diphenyl-1,6,2,3-tetrahydropteridine (VI) (I, R, R' = Ph), the **oxidation** product (VII) of VI, 2-ethylmercapto-6-hydroxy-8,9-diphenylpteridine (VIII) (II, R, R' = Ph), and 2,6-dihydroxy-8,9-diphenylpteridine (IX) (IV, R, R' = Ph) are detd. I and VI show absorption max. at 3065 and 3180 A. (Hg light). On **oxidation** of I and VI, the absorption max. shift to the shorter wave lengths, those of V, II, and IV lying at 2700, 2715, and 2700 A., resp., those of VII, VIII, and IX at 2870, 2840, and 2870, resp. The fluorescence of the compds. with R, R' = H is very strong in alk. soln. and becomes very weak in acid soln. due to the change from the lactim form into the lactam form under the influence of the pH. Since some S compds. exhibit an antifluorescence activity (cf. Perrin, C.A. 21, 3012), it is possible that a fluorescence which may originate from an equil. between the thione form (X) and the thiol form (XI) in I may be prevented by the anti-fluorescence properties of X. The effect of thiourea (XII) upon the fluorescence of II (R, R' = H) and III is studied, and it is found that XII, unlike urea, exhibits a strong antifluorescence activity. Because I contains the same grouping as is present in XII, P. et al. assume that I also acts as a fluorescence **inhibitor**. Addn. of I to a soln. of II or III also decreases the fluorescence of the latter to an even greater extent than the addn. of XII.

L9 ANSWER 69 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1942:2977 CAPLUS
DOCUMENT NUMBER: 36:2977
ORIGINAL REFERENCE NO.: 36:508b-d
TITLE: Using Phycomyces blakesleeanus in the determination of vitamin B1
AUTHOR(S): Malm, Max; Lundeen, Harry
SOURCE: Svensk Kem. Tid. (1941), 53, 246-64
DOCUMENT TYPE: Journal
LANGUAGE: German
AB cf. Sinclair, C. A. 33, 4625.8. Vitamin B1 is detd. by the increase in dry wt. of the mycelium of Phycomyces in glucose-asparagine substrate to which the sample has been added. Casein hydrolyzate, age of culture from which spores are taken, and light are **inhibiting** factors of growth but do not interfere sufficiently to vitiate the method. Equiv. concns. B1, cocarboxylase, B1 orthophosphate, disulfide and **pyrimidine** + thiazoleacetate from B1 have the same effect on the growth. The disulfide of B1 gives a slower growth rate than B1 and the rate increases with the concn. of the disulfide. **Pyrimidine** sulfonate + thiazole, nicotinamide, adermin, .beta.-alanine, cystine, glutathione and heparin do not promote the growth of the fungus. Thiochrome from B1 by $K_3FeC_6N_6$ **oxidation** does not promote growth. In an ideal substrate the increased mycelium wt. is directly proportional to the concn. B1. A method of calcn. is given for substrates having activating or **inhibitory** factors. Sinclair's correction

factor is not accepted.

L9 ANSWER 70 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1941:48047 CAPLUS
DOCUMENT NUMBER: 35:48047
ORIGINAL REFERENCE NO.: 35:7455g-i,7456a-c
TITLE: Vitamin B1 and bacterial oxidations. I. Dependence of acetic acid **oxidation** on vitamin B1
AUTHOR(S): Quastel, J. H.; Webley, D. M.
SOURCE: Biochem. J. (1941), 35, 192-206
DOCUMENT TYPE: Journal
LANGUAGE: Unavailable
AB cf. C. A. 34, 791.5. The presence of vitamin B1 (I) at low concns. (10.-7 M) greatly increased the rate of **oxidation** of AcOH by I-deficient propionic acid bacteria. The molar ratio of O consumed to AcOH used in the presence and absence of I was approx. 2.0; this shows complete **oxidation** of AcOH. HCO2H, AcOH, EtCO2H and PrCO2H were oxidized at the same rate by I-deficient propionic acid bacteria. The addn. of I caused little change in the rate of **oxidation** of HCO2H and PrCO2H, but increased the rate of **oxidation** of EtCO2H. The increase was much less than with AcOH. Pyruvic acid (II) accumulated during the **oxidation** of glucose (III), lactic acid (IV), glycerol (V) and PrCO2H by deficient bacteria. Its amt. was greatly decreased in the presence of I. The rate of utilization of AcOH by these organisms with added I was approx. equal to that of II. The presence of IV, succinic acid (VI), fumaric acid (VII) or V **inhibited** the utilization of II by propionic acid bacteria in the presence of I. EtCO2H caused some **inhibition** but AcOH had no effect. The presence of I greatly stimulated the rates of respiration by deficient bacteria with the following substrates: II, IV, VI, VII, III, V. Definite but less marked effects were shown by I in the oxidations of l-malic acid, l-glutamic acid, dl-alanine, glycolic acid, glycine and fructose. Little or no **oxidation** occurred in any case with oxalic acid, citric acid or .beta.-hydroxybutyric acid. The **oxidation** of .alpha.-glycerophosphoric and hexosediphosphoric acids was not affected by I. The stimulant action of I was attributed mainly to its effect on the **oxidation** of II formed as an intermediate. The effect of I on the **oxidation** of AcOH could not be explained in this way. EtOH was oxidized by I-deficient propionic acid bacteria, the rate of **oxidation** being greatly increased by the addn. of I. AcOH which accumulated during the **oxidation** of EtOH was greatly reduced when I was present, probably accounting for the increased rate under such conditions. PrOH was vigorously oxidized by deficient bacteria, but the rate was not affected greatly by I. MeOH was only slightly oxidized in both instances. The **pyrimidine** and thiazole components of I were separately unable to catalyze the **oxidation** of AcON by propionic acid bacteria.

L9 ANSWER 71 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1938:39030 CAPLUS
DOCUMENT NUMBER: 32:39030
ORIGINAL REFERENCE NO.: 32:5437a-b
TITLE: Aneurin (vitamin B1) and pyruvate metabolism by Staphylococcus aureus
AUTHOR(S): Hills, Geo. M.
SOURCE: Biochem. J. (1938), 32, 383-91
DOCUMENT TYPE: Journal
LANGUAGE: Unavailable
AB The effect of aneurin (I) on the metabolism of Staph. aureus occurred at very low concns. and could only be observed in organisms which received an inadequate supply of the vitamin during growth. Both the **pyrimidine** and the thiazole rings of I were necessary for the normal metabolism of pyruvic acid (II) by staphylococcus both under

aerobic and anaerobic conditions. In the metabolism of lactate (III) and II the presence of I was necessary for the reaction which involved the dismutation of I into lactic acid, AcOH and CO2. In the absence of I, there was an accumulation of II which acted as an **inhibitor** on the **oxidation** of III.

L9 ANSWER 72 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1937:38292 CAPLUS
DOCUMENT NUMBER: 31:38292
ORIGINAL REFERENCE NO.: 31:5395c-f
TITLE: Decomposition of histidine and of other imidazoles by ascorbic acid
AUTHOR(S): Edlbacher, S.; v. Segesser, A.
SOURCE: Biochem. Z. (1937), 290, 370-7
DOCUMENT TYPE: Journal
LANGUAGE: Unavailable

AB Histidine unlike other amino acids is absolutely resistant to oxidative deamination but its imidazole ring is split open hydrolytically by a histidase present in the liver. There is a close analogy to the related arginine which is likewise hydrolyzed to ornithine and urea by liver arginase. The histidase gives a labile product contg. one N atom less, which on alkalinizing with NaOH gives off a second N atom as NH3, and acts equally well in the presence or absence of O2 and is unaffected by KCN. When histidine is incubated with ascorbic acid at pH 7 in the presence of a trace of Fe2(SO4)3 or of hemin similar products are formed, only in this case there is an oxidative reaction (depends on presence of O2 and is **inhibited** by CN). Expts. with various biologically important substances show that only the simple imidazole derivs. are destroyed by this reaction whereas hypoxanthine and adenine with the imidazole ring bound to a **pyrimidine** ring are at least partially deaminated. The question whether other reduction-**oxidation** substances besides ascorbic acid can be used is being investigated. The biol. significance of the place occupied by ascorbic acid in the **oxidation**-reduction system of the cell is discussed.

L9 ANSWER 73 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1935:19870 CAPLUS
DOCUMENT NUMBER: 29:19870
ORIGINAL REFERENCE NO.: 29:2554a-c
TITLE: Experimental studies on nuclein metabolism. XXXVII. Nucleosidase
AUTHOR(S): Klein, Willibald
SOURCE: Z. physiol. Chem. (1935), 231, 125-48
DOCUMENT TYPE: Journal
LANGUAGE: Unavailable

AB cf. C. A. 28, 4079.9. A micro method for detg. hydrolysis of purine desoxyribosides by nucleosidase is based on removal of protein by Pb(NO3)2, addn. of 0.02 N I and 0.1 N NaOH, acidification with N H2SO4 and titration of excess I with 0.01 N Na2S2O3 and starch indicator. The intact nucleosides are not attacked but the liberated desoxypentose undergoes **oxidation**. Nucleosidase is obtained by drying frozen organs, adsorbing the ext. on Al(OH)3 and eluting with Na2HAsO4, the most active prepns. being obtained from spleen, lung, liver and heart muscle. AsO4---, and to a smaller extent PO4---, is the activator. The enzyme is sp. for the purine nucleosides and does not attack **pyrimidine** nucleosides, nuclecotides or nucleic acids. Guanine and hypoxanthine are strong **inhibitors**, adenine is less so and desoxyribose very slightly so, while the **pyrimidine** nucleosides and the nucleotides have no effect. Purine nucleosidases from various organs are identical, as are also the riboside and desoxyriboside nucleosidases. In addn. to purine nucleosidase there is a sp. **pyrimidine** nucleosidase. This is more abundant in kidney than in spleen and red marrow.

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     FILE 'REGISTRY' ENTERED AT 17:22:55 ON 02 SEP 2002
L1             STRUCTURE UPLOADED
L2               3 S L1
L3             330 S L1 FUL

     FILE 'CAPLUS' ENTERED AT 17:23:39 ON 02 SEP 2002
L4             323 S L3
L5            9814 S ELECTRON DONATING
L6               1 S L4 AND L5
L7           24310 S INHIBIT? AND OXIDATION
L8               0 S L4 AND L7
L9              73 S L7 AND PYRIMIDIN?
L10             14 S L9 AND HYDROXY
L11              0 S L7 AND (HYDROXY SAME PYRIMIDIN?)
L12              0 S L7 AND (HYDROXY PYRIMIDIN?)

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